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VOLUME 33 NUMBER 10 CALCUTTA NOVEMBER 16, 1959

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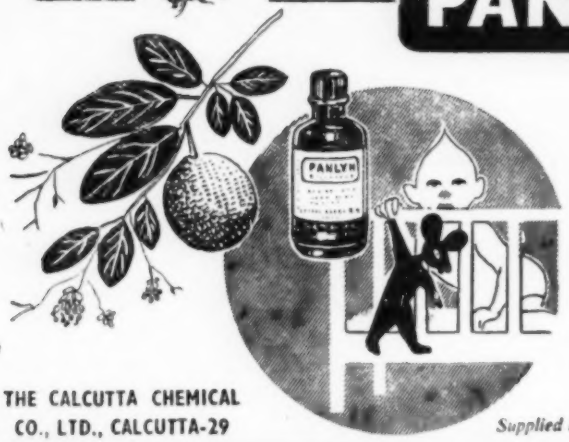

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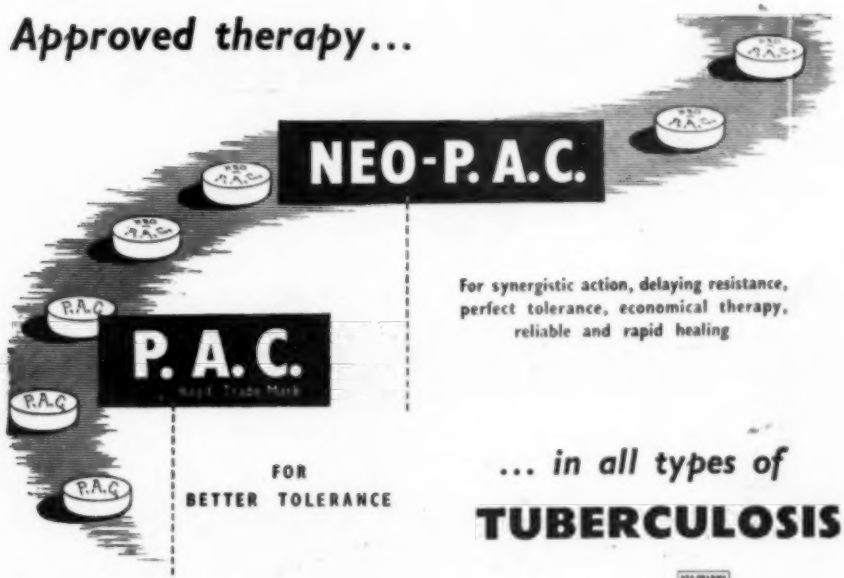

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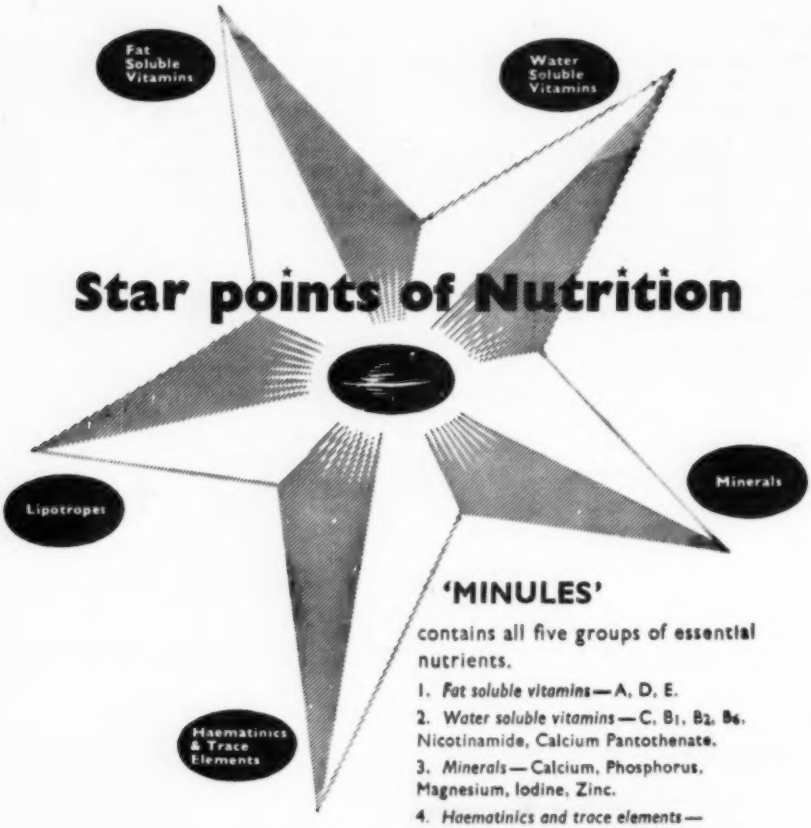

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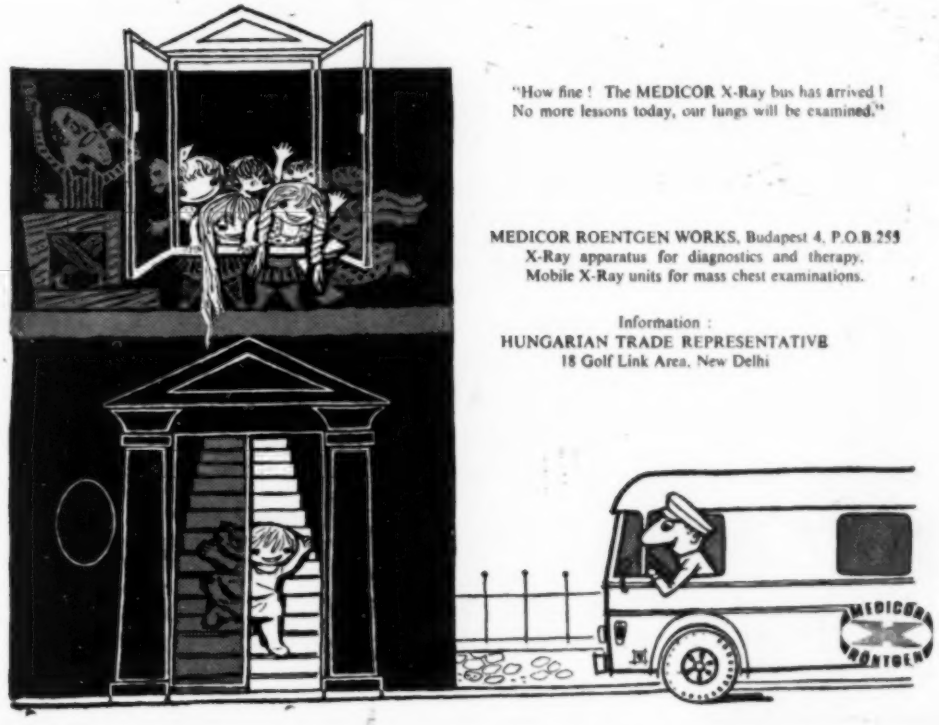

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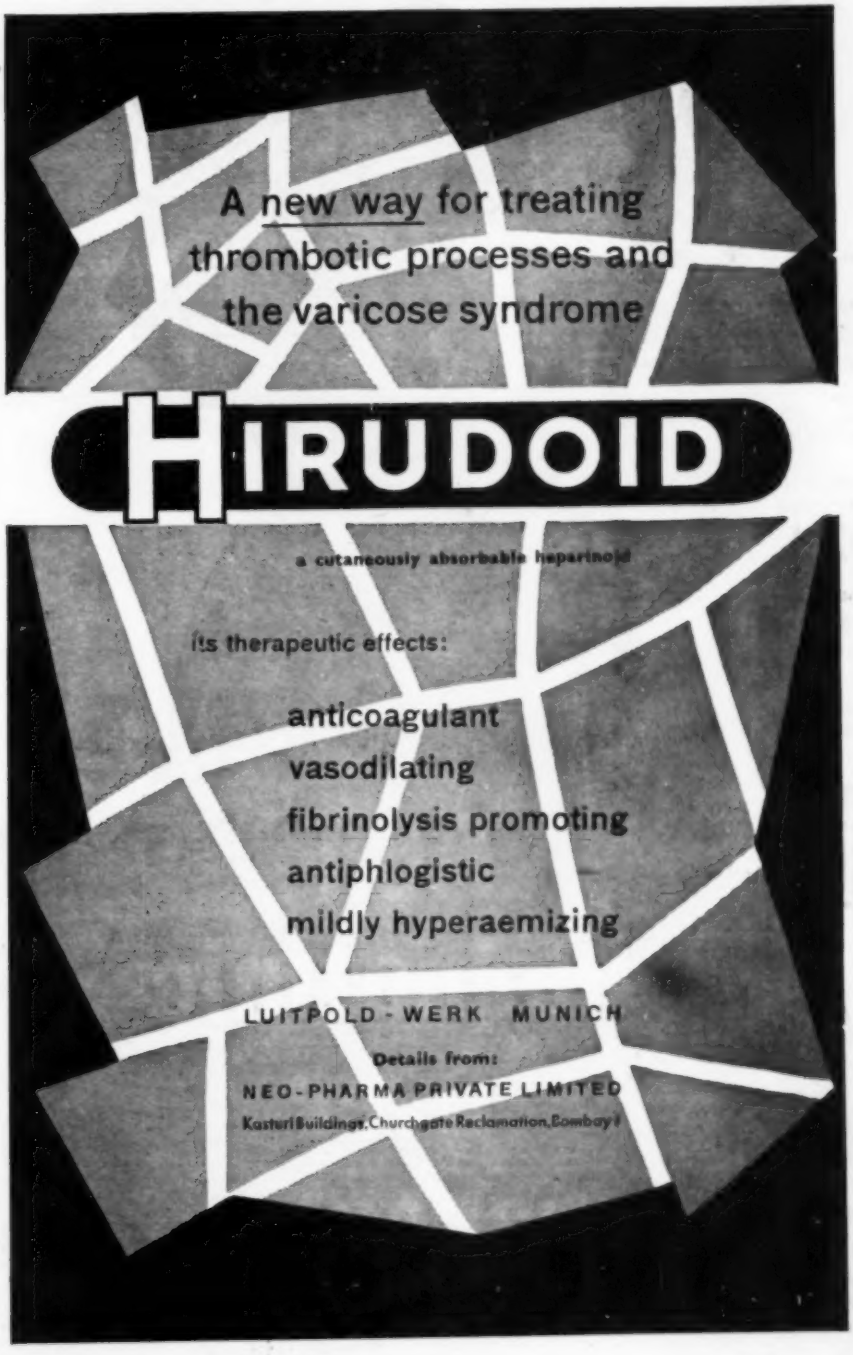
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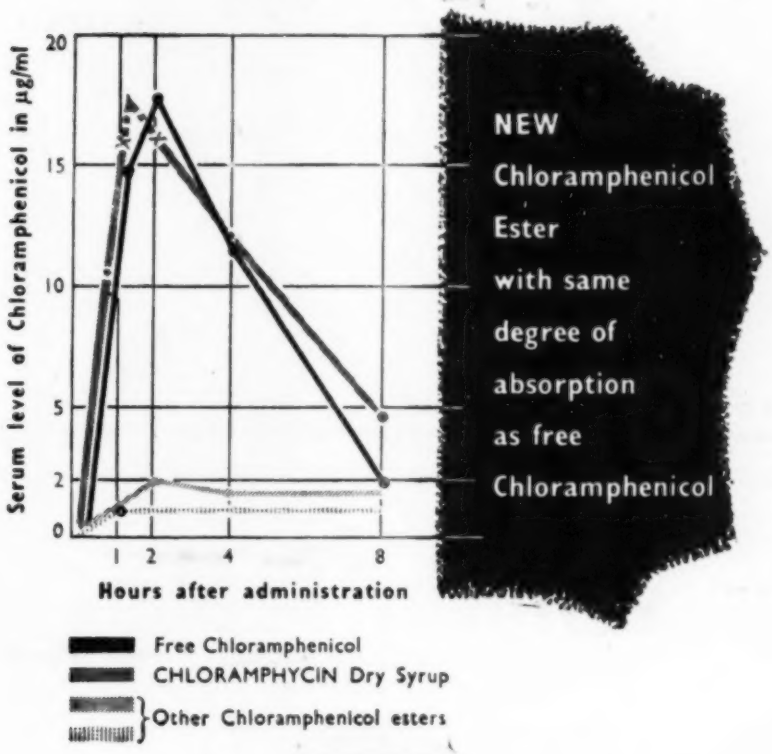

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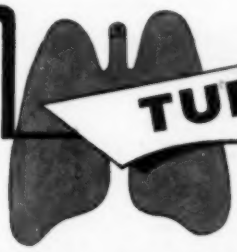

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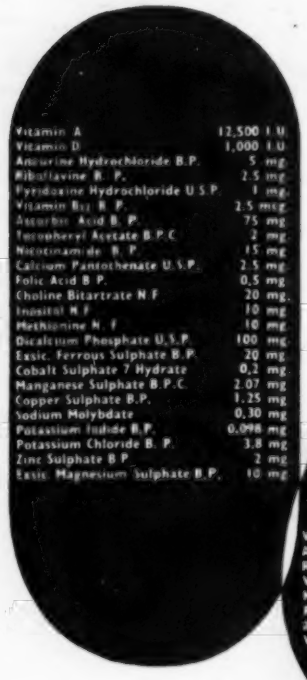
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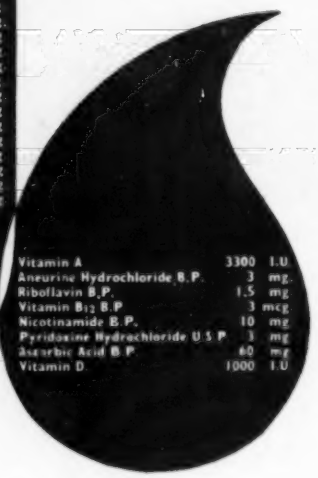
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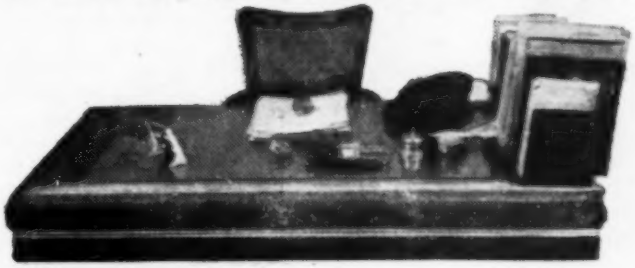

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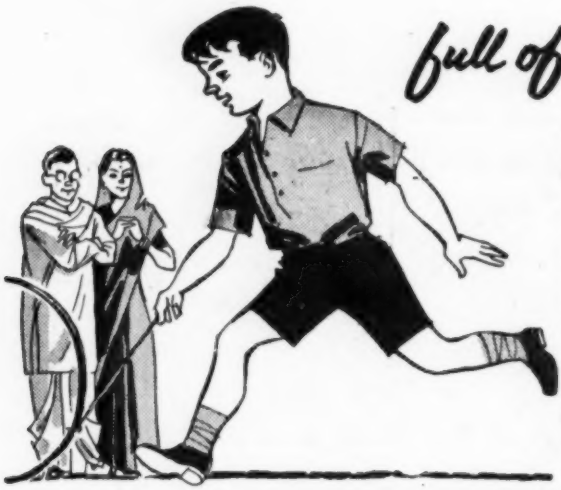

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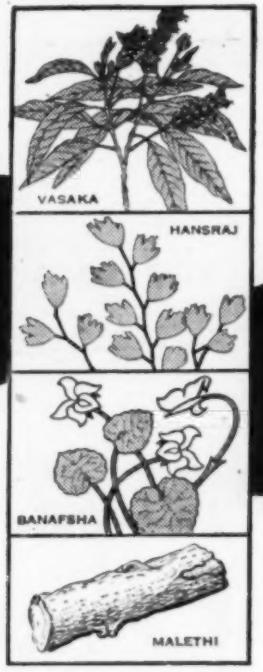

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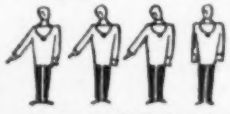

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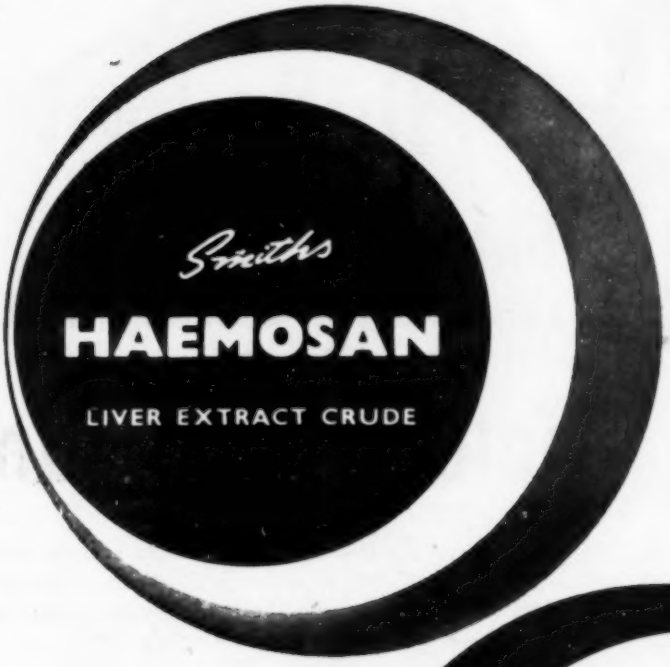

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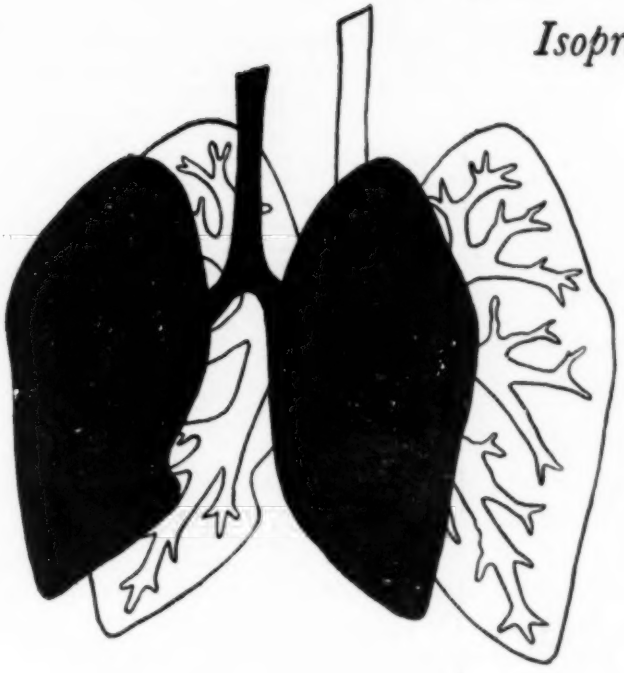

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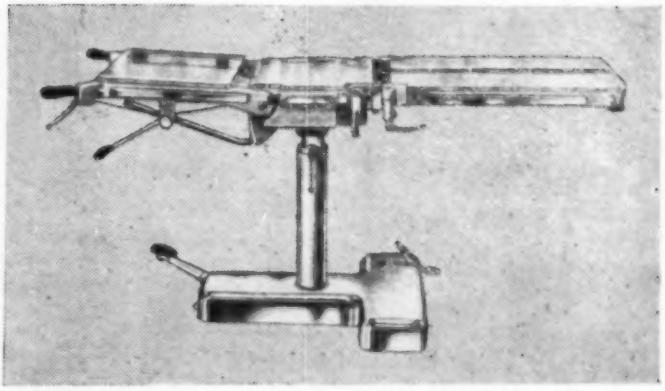

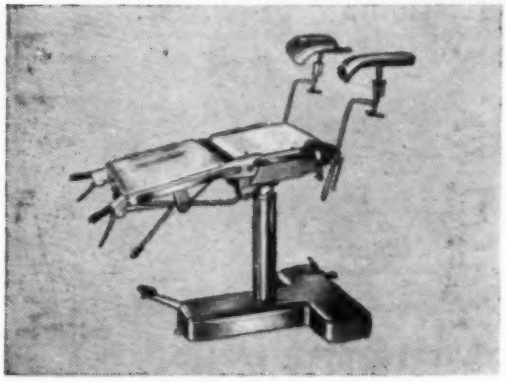

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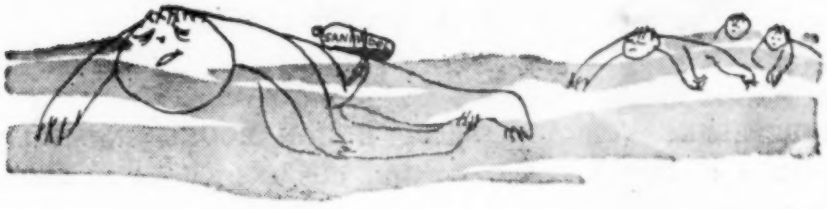

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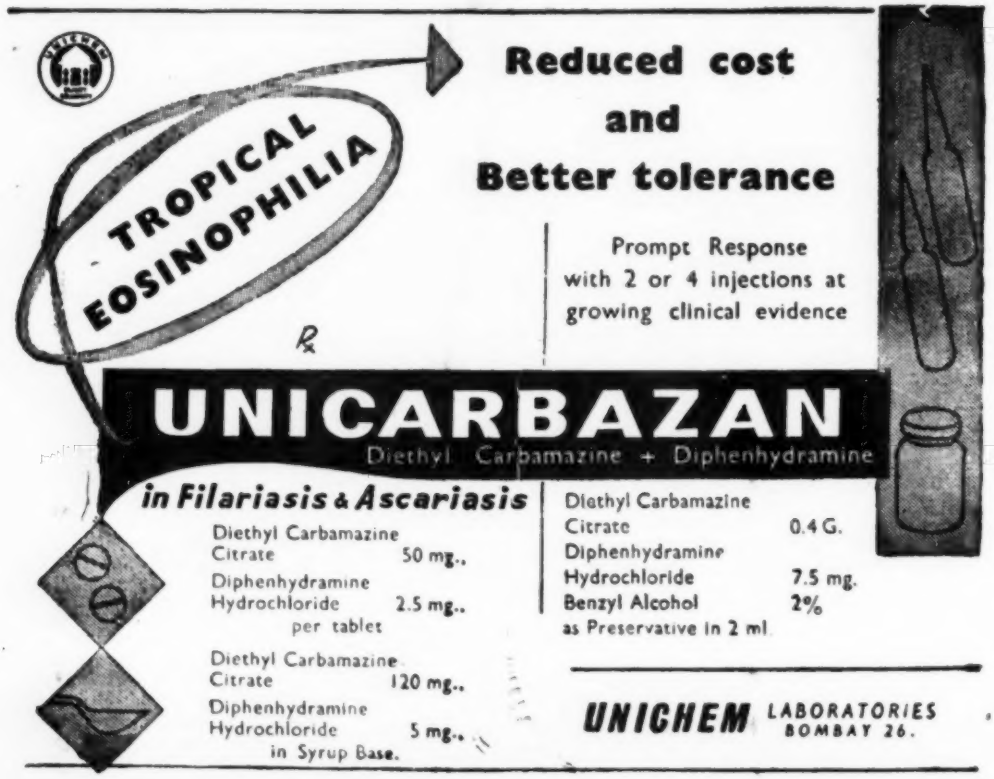
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Journal of the Indian Medical Association

VOLUME 33
NUMBER 10

PUBLISHED TWICE A MONTH
EDITOR—P. K. GUHA, M.B., M.R.C.S. (ENG.), D.O.M.S. (LOND.)

CALCUTTA
NOVEMBER 16, 1959

ORIGINAL ARTICLES

STUDY OF BITOT'S SPOT WITH SPECIAL REFERENCE TO PROTEIN MALNUTRITION

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Bitot's spot is recognised as a manifestation of vitamin A deficiency in man. Since Bitot first described this condition in 1865, many workers from different parts of the world (Wolbach and Howe, 1922 ; Nicholls and Nimalasuriya, 1939 ; Aykroyd and Rajagopal, 1936-37) confirmed this eye sign to be a manifestation of vitamin A deficiency. In addition, detailed observations were also made by these workers on its clinical features. At present, it is used as a clinical sign for evaluating vitamin A status so that its presence in a subject indicates vitamin A deficiency. Histologically, Bitot's spot consists of piled up keratinised cells. Scrapings taken from these patches show desquamated keratinised cells. The histological features corroborate the aetiology of Bitot's spot.

Nutritionists, working in areas where protein malnutrition is rampant, e.g., South-East Asian countries are gradually becoming aware of the possibility of protein malnutrition playing some part in precipitating or aggravating vitamin A deficiency, especially in relation to eye lesions. The Nutrition Committee for South and East Asia, under the auspices of FAO and WHO in their Fourth Session at Tokyo in 1956 also emphasised this fact. They observed that a considerable proportion of the cases of protein malnutrition described in India and Indonesia had been reported to show signs of avitaminosis A. The Joint FAO/WHO Expert Committee on Nutrition (1958) in their Fifth Report states that the association of protein malnutrition and xerophthalmia or keratomalacia indicates that the diet lacks both in vitamin A activity and protein. Yap-Kie-Tiong (1956) supposes that keratomalacia, which is regarded as a severe manifestation of vitamin A deficiency in the eyes, is more closely dependent on protein malnutrition. Oomen (1958) draws a line of distinction between xerophthalmia and keratomalacia. He thinks keratomalacia is a colliquative necrosis quite distinct from xerophthalmia. However, both are serious conditions as they can produce permanent blindness. Success of vitamin A therapy is often not great in keratomalacia.*

* 14 per cent of cases of vitamin A deficiency shows Bitot's spots and 21 per cent shows pigmentation. Cases of keratomalacia make very rapid improvement often by 48-96 hours after parenteral administration of vitamin A (100,000 units).—ED.—*J. Indian M. A.*

Considering these factors, it was realised that an investigation on Bitot's spot and its clinical response to different types of therapy might yield some solution as to what are the factors operating in the genesis of this condition.

In the Nutrition Clinic of the Urban Health Centre of the All-India Institute of Hygiene and Public Health, 46 cases of Bitot's spot were closely studied and investigated both clinically and biochemically before and after treatment with different nutrients during which some new observations were made regarding the clinical features, actiology and treatment of this condition.

Clinical Features

Pigmentation—For a long time pigmentation of the sclera has been a source of controversy regarding its connection with nutritional deficiency and with Bitot's spot. Brownish or muddy discolouration of the sclerae, either generalised or in patches has been regarded by several workers in the past to be a manifestation of vitamin A deficiency but none of these workers are definite about it since no careful study has been made so far in this respect. Bitot's spot has also been observed to be associated with pigmentation but no emphasis has been placed on this so that the association between pigmentation and Bitot's spot does not, uptil now, seem to be significant.

Pigmentation, on the other hand, has been found in this investigation to be of great importance. In 6 subjects, the Bitot's spot was found to be unilateral but on careful examination, it was observed that in the other eye, a comparable area was found to be pigmented with a brownish to chocolate tint. With treatment, the pigmented triangular patch started clearing up before any improvement in the Bitot's spot was observed, which, however, took a very long time to clear up. In a considerable number of children, who were being treated for protein malnutrition, pigmented patches were observed in both the eyes in areas which are usually occupied by Bitot's spot. These pigmented patches usually cleared up with protein therapy. Those not treated, gradually deteriorated to Bitot's sport.

The pigmented patch, before its conversion to the characteristic Bitot's spot, occupies the same area as that of its successor. On careful examination, it was found to be slightly thickened but the surface was not so raised as is usually seen in Bitot's spot. The surface though slightly wrinkled has not the characteristic appearance of Bitot's spot. Another marked difference from Bitot's spot is the presence of a number of engorged capillaries in the area. When these pigmented patches gradually progress towards full-fledged Bitot's spot, the sequence of changes is: disappearance of capillaries and diminution of pigmentation leaving a yellowish patch over which the non-transparent keratinised cells appear, giving a characteristic appearance, which Aykroyd and Rajagopal (1936-37) have described as "yellowish foamy patches".

Capillary distribution—The distribution of capillaries in the pigmented area was also found to be quite characteristic. A single engorged capillary leaves the outer canthus and extends along the equator of the eye towards the cornea. Midway, it breaks up into several branches which reach the limbus almost in a triangular fashion. This triangular patch becomes pigmented and later on undergoes keratinisation forming Bitot's spot.

Other associated nutritional deficiencies—Table 1 gives a description of associated nutritional deficiencies in 46 cases of Bitot's spot investigated in the present work.

Table 1—Showing Manifestations of Associated Nutritional Deficiencies

Manifestations of associated deficiency	Probable deficiency	No. of cases
Nightblindness	Vitamin A	4
Angular stomatitis and glossitis	Riboflavin	2
Enlarged liver, relative lack of growth, brownish discolouration of hair with inadequate muscle mass and hypotonia	Protein	40

A careful consideration of Table 1 poses two important questions:

(1) If Bitot's spot is purely due to a deficiency of vitamin A, as is now accepted, it is surprising to note the absence of other associated manifestations of vitamin A deficiency with Bitot's spot. For example, in the present series of 46 cases, only 4 had associated nightblindness which is regarded as a manifestation of vitamin A deficiency.

(2) Another surprising fact is the very large percentage of these cases showing evidences of protein malnutrition. It is quite pertinent to enquire whether this observation can be explained as due to an associated protein deficiency which is so common amongst the population or that protein malnutrition plays some part in precipitating or aggravating Bitot's spot.

Biochemical Investigation

Vitamin A was determined in the serum of all the subjects before starting any treatment according to the method of Bessey *et al* (1946). Serum protein was determined by microkjeldahl method. The albumin and globulin fractions were determined by quantitative method of King and Wootton (1956) based on biuret reaction. Liver function tests were also done to assess the functional capacity of the liver which plays an important role in the storage and distribution of vitamin A and also due to its close link with protein malnutrition. The two tests performed in every case were thymol turbidity test (Shank and Hoagland, 1946) and zinc sulphate turbidity test (Kunkel, 1947).

Table 2—Showing the Results of Biochemical Investigations

Case No.	Sex & Age in years	Plasma vitamin A (γ/100 ml.)	Total plasma protein (g./100 ml.)	Albumin (g./100 ml.)	A : G ratio	Thymol turbidity (units)	Zinc sulphate turbidity (units)
1.	M, 5	17·0	5·8	3·7	1·8	9·2	15·6
2.	M, 2-8/12	11·7	5·6	3·7	1·9	7·8	15·2
3.	M, 4-6/12	12·8	5·6	3·8	2·1	7·2	13·8
4.	F, 6-2/12	13·4	6·4	4·5	2·3	7·6	13·8
5.	M, 5	12·5	5·3	3·4	1·7	7·8	14·2
6.	F, 10	18·5	5·6	3·7	1·9	8·4	16·2
7.	F, 8	12·6	5·0	2·7	1·2	13·6	19·2
8.	M, 12	14·7	5·1	3·0	1·4	11·8	18·8
9.	F, 4-6/12	15·3	5·8	3·5	1·6	9·6	17·8
10.	M, 7-3/12	8·7	4·9	2·6	1·1	15·8	23·2
11.	F, 8-6/12	15·3	5·1	2·9	1·3	13·4	4·6
12.	M. 6-2/12	12·0	4·8	2·5	1·1	11·6	19·4
13.	F, 5-3/12	10·8	5·6	3·4	1·5	11·8	20·4
14.	M, 3-4/12	9·3	4·9	2·6	1·1	15·2	23·6
15.	M, 4-6/12	12·6	5·8	3·7	1·8	13·4	21·8
16.	F, 6-3/12	11·1	5·2	2·9	1·2	11·8	19·6
17.	M, 4-6/12	9·3	5·4	2·8	1·1	15·2	21·8
18.	F, 5-7/12	8·7	5·0	2·8	1·2	13·6	19·6
19.	F, 3-7/12	10·1	5·8	3·7	1·7	15·4	21·6
20.	F, 4-3/12	12·0	5·7	3·5	1·6	11·6	19·2
21.	M, 5-7/12	10·5	4·8	2·5	1·1	15·8	23·6
22.	F, 4-8/12	12·3	5·6	3·5	1·6	13·4	21·8
23.	M, 18	9·3	5·6	3·0	1·1	11·6	21·2
24.	F, 10	12·3	5·6	3·5	1·6	9·8	17·4
25.	F, 8	8·7	4·8	2·4	1·0	17·6	25·8
26.	M, 6-3/12	10·8	5·6	3·7	1·8	11·8	19·6
27.	F, 4	13·8	5·7	3·7	1·9	8·4	19·2
28.	M, 5-6/12	12·3	6·1	3·8	1·6	11·6	21·8
29.	F, 4	9·6	5·8	3·0	1·1	13·8	23·6
30.	F, 5-6/12	10·8	5·4	3·0	1·2	11·6	19·2
31.	F, 4-6/12	14·4	5·2	3·1	1·4	11·8	29·2
32	M, 6-7/12	11·1	5·8	3·3	1·3	13·6	21·6
33.	F, 7	9·3	5·1	2·7	1·1	10·8	19·4
34.	F, 6-7/12	10·2	5·4	3·0	1·2	13·8	23·6
35.	M, 3-6/12	12·3	5·6	3·3	1·4	11·6	19·2
36.	F, 4	14·7	5·4	3·5	1·9	12·8	21·8
37.	M, 6-6/12	12·6	5·1	2·9	1·4	11·6	19·6
38.	F, 5	11·1	5·6	3·1	1·2	13·6	18·8
39.	F, 10-6/12	10·2	5·1	2·7	1·1	10·2	17·8
40.	M. 4	8·1	4·9	2·5	1·0	15·6	27·6
41.	F, 5-3/12	10·8	5·2	2·9	1·2	11·6	19·6
42.	F, 6	12·3	5·6	3·2	1·3	11·8	22·4
43.	M, 4-6/12	11·1	5·1	2·9	1·3	15·6	25·2
44.	F, 6	14·7	5·8	3·3	1·3	13·8	23·2
45.	M, 7	12·6	5·4	3·1	1·3	15·2	25·6
46.	F, 4-6/12	11·1	5·1	2·7	1·1	13·6	25·6

Though the number of cases of Bitot's spot associated with signs other than those of protein malnutrition is quite small, yet it is fairly clear from the biochemical values (Table 2) that the serum vitamin A level and A : G ratio in these cases are considerably different from similar values in cases associated with signs of protein malnutrition. Liver function tests gave higher values in all the cases of Bitot's spot having manifestations of protein malnutrition.

Statistical Analysis

The group is composed of 20 boys and 26 girls. Large majority (30) of the children belong to the age group 4-6. The average standard deviations (S. D.) and co-efficients of variation (C. V.) of the measures of the biochemical constituents were as follows:

	Average	S.D.	C.V.
Plasma vitamin A ...	11·86	2·24	18·9
Total protein ...	5·41	0·37	6·8
Albumin	3·17	0·46	14·5
Thymol turbidity ...	12·27	2·51	20·5
Zinc sulphate turbidity ...	20·06	3·98	19·8

Association of the levels of vitamin A with those of protein and albumin—Vitamin A was found to be positively correlated with albumin, the co-efficient of correlation being + 0·505 with the rest of protein, i.e., total protein minus albumin. The observed correlation between vitamin A and albumin may, therefore, arise either partly or wholly due to the variation of the remaining part of total protein. This question may be decided by working out the partial correlation co-efficient between vitamin A and albumin keeping the rest of the protein constant. This partial correlation co-efficient works out to + 0·354 and is statistically significant. Hence even after taking into consideration the variation in the levels of the

TABLE 3—SHOWING THE RESULTS OF BIOCHEMICAL TESTS AFTER TREATMENT

Case No.	Mode of treatment	Duration of treatment (weeks)	Clinical response	Plasma vitamin A (γ/100 ml.)	Plasma protein (g./100ml.)	Albumin (g./100ml.)	A : G ratio	Thymol turbidity (units)	$ZnSO_4$ turbidity (units)
1.	Vitamin A	26	Slight improvement	47·4	6·4	4·2	1·8	8·2	16·2
2.	Vitamin A	24	,,	42·3	5·6	3·6	1·8	7·4	15·8
3.	Vitamin A and protein	24	Almost cured	64·8	6·3	4·4	2·3	5·6	13·4
4.	Vitamin A and protein	28	Cured	58·8	6·6	4·8	2·6	7·4	12·8
5.	Vitamin A (Riboflavin given for glossitis and stomatitis)	20	No improvement	43·2	5·1	3·2	1·6	7·6	15·2
6	Vitamin A and protein (Riboflavin given for glossitis and stomatitis)	21	Marked improvement	67·8	6·2	4·2	2·1	6·4	15·6
7.	Vitamin A and protein	24	Cured	59·4	5·9	3·9	1·9	8·2	15·2
8.	Vitamin A	27	Slight improvement	35·1	5·0	2·9	1·3	12·2	21·2
10.	Vitamin A	18	No improvement	32·4	5·0	2·7	1·1	15·2	24·4
12.	Vitamin A and protein	18	Marked improvement	55·8	5·6	3·6	1·8	8·4	17·8
14.	Vitamin A	20	Slight improvement	43·8	4·9	2·7	1·2	15·6	23·2
16.	Vitamin A and protein	19	Almost cured	65·1	5·9	3·9	2·0	7·2	14·4
18.	Vitamin A	20	No improvement	32·4	5·1	2·7	1·1	13·4	21·2
21.	Vitamin A and protein	19	Almost cured	60·3	5·9	3·9	1·9	7·2	15·6
23.	Vitamin A	24	Good improvement	50·4	5·6	3·2	1·3	11·8	21·6
24.	Vitamin A and protein	25	Cured	63·0	6·4	4·4	2·2	5·6	11·2
26.	Vitamin A	24	Moderate improvement	53·4	5·7	3·7	1·9	10·4	16·1
30.	Vitamin A and protein	24	Cured	64·8	6·2	4·2	2·1	7·2	14·4
31.	Vitamin A	22	Slight improvement	32·7	5·1	2·9	1·3	12·6	20·6
33.	Vitamin A and protein	22	Cured	59·1	5·9	3·9	1·9	5·4	13·6
37.	Vitamin A	18	Slight improvement	34·8	5·0	2·7	1·2	12·2	15·6
39.	Vitamin A and protein	20	Cured	56·1	6·0	3·9	1·9	5·6	7·8
40.	Vitamin A	22	No improvement	30·3	4·9	2·5	1·0	15·2	26·4
41.	Vitamin A and protein	21	Almost cured	63·0	6·1	4·1	2·1	3·6	6·8
43.	Vitamin A	24	Slight improvement	33·6	5·0	2·6	1·1	13·4	22·4
46.	Vitamin A and protein	23	Cured	54·6	5·9	3·9	1·9	5·4	11·6

Slight improvement —Diminution in the size of the patch and also in keratinisation.
Marked improvement —Almost complete disappearance of surface keratinisation and aluminium paint flakes. Surface of the patch gets flushed with the general surface of sclera.
Almost cured —Brownish patchy pigmentation in the area occupied by Bitot's spot.

rest of protein, vitamin A is observed to be positively correlated with albumin.

Response to vitamin A and protein—Considering the biochemical evidence of a possibility of protein malnutrition playing a part in the aetiology of Bitot's spot, it was decided to treat these cases by two different methods. One group was treated with vitamin A acetate (Arovit) liquid orally, 50,000 I.U. daily. The patients were made to come to the Nutrition Clinic where they had to take the vitamin supplement in presence of the staff. The other group was treated with the same vitamin in exactly the same amount and similar precautions were taken to ensure that the correct amount of the supplement was being taken daily, but, in addition, the patients were given skimmed milk powder in such amount that each patient consumed 32 oz. of reconstituted skimmed milk daily yielding about 30 g. of protein. Frequent visits by the dietitian and other field staff served as check. In most cases, the supplementation of 32 oz. milk to their usual diet did not cause any dislocation of their dietary pattern. In a small number of cases, the amount of rice had to be reduced in order to enable the children to consume milk.

The eyes were clinically examined once a week to watch the effect of such treatment. Blood was collected during the course of treatment and biochemical examination, mentioned before, done to determine the effect of treatment.

Clinical Response

With vitamin A alone—Treatment with vitamin A alone, as described above takes a very long time to give clinical response. Two cases of Bitot's spots with nightblindness, which presumably are cases of pure vitamin A deficiency, also behaved in the same manner though the nightblindness improved considerably. Previous workers in this line have also reported the same experience in treating Bitot's spot. On the average, it took about 24-28 weeks on this dosage to get some clinical response.

With vitamin A and protein—This form of treatment gave surprisingly quick clinical response. Even the two cases, associated with nightblindness and supposed to be due to purely vitamin A deficiency showed a very rapid improvement with this form of treatment. Subjects with signs of protein malnutrition, however, showed striking improvement, not only in their eye signs but also a very marked general improvement due to protein supplement. Biochemically, the most marked change as related to clinical improvement was seen in the results of liver function tests. The average time taken for regression to the pigmented stage was 5-6 weeks and by 16-20 weeks the condition almost disappeared.

Biochemical Response

The biochemical examinations which were done before starting the treatment were again repeated when fairly satisfactory clinical response was obtained. Blood was collected only from 28 subjects, the remaining patients having refused to give blood for the second time. Table 3 gives the result of these biochemical examinations. The case numbers given in Table 3 are the same as those in Table 2 and as such biochemical values before starting the treatment can be easily obtained for the sake of comparison by referring to Table 2.

Discussion

The clinical response given by the two different types of treatment show beyond doubt that vitamin A given with about 30-32 g. of protein daily has a far better curative effect on Bitot's spot. Surprisingly, a liberal dose of vitamin A orally for a period varying from 18 to 28 weeks failed to completely cure this condition in some cases whereas the same dose supplemented with a good amount of milk protein produced a very marked response.

This observation poses some very important questions. Can Bitot's spot be described as a deficiency of vitamin A superimposed on protein malnutrition? The current trend of thought is that it is entirely due to vitamin A deficiency and no other contributory factor has been mentioned so far. A reference to literature on this subject would prove that it has always been seen in malnourished communities, especially in children and it is extremely difficult to treat with the missing vitamin. All these factors would support the assumption that protein malnutrition plays a significant part in its aetiology.

Several possible explanations can be advanced for this role of protein:

(a) Protein malnutrition damages the pancreas even in its very early phase (Davies, 1948; Véghelyi, 1948). A damaged pancreas, especially when its acinar parts are involved, may so affect the digestive enzymes that though vitamin A might be present in adequate amount in the diet, the amount absorbed would be greatly reduced. Inadequate digestion and absorption of fat might also seriously impair the absorption of vitamin A and carotene.

(b) Pathological changes in the intestinal wall might adversely affect the absorption of vitamin A and carotene and also the conversion of carotene to

vitamin A. Stransky (1950) has demonstrated atrophy of the mucosa of the gastro-intestinal tract in kwashiorkor.

(c) Damaged liver, an almost constant finding in protein malnutrition and kwashiorkor (Anderson and Altmann, 1951 ; Gillman and Gillman, 1945 ; Himsworth, 1950) might impair the storage and utilisation of vitamin A and carotene.

(d) Vitamin A in serum has been found to be in some form of combination with protein, probably albumin. It is well known, that in protein malnutrition, the serum protein level is lowered but much more specific is the lowering of albumin fraction (Trowell, 1950). It is probable that this decrease of the carrier of vitamin A in blood might affect its supply to different tissues. A reference to Table 3 will throw some light on these points. The uniformly low vitamin A level in the serum of the patients treated with vitamin A alone is quite significant when compared to similar value in patients treated with vitamin A and protein. This fact might be explained as due to defective digestion *cum* absorption or due to decreased capacity of the serum to carry vitamin A due to hypo-albuminaemia.

(e) There is almost a complete gap in our knowledge of the cellular metabolism of vitamin A. Excepting its specific role in the retina, nothing is known as to how exactly this vitamin functions in the body (Handler, 1958). Keratinisation, the most commonly described effect of vitamin A deficiency on the epithelium, has not even been explained satisfactorily on the basis of its biochemical and metabolic role. Whatever work has been done in recent years on these lines points to the interdependence of vitamin A and SH group, which depends on protein for activity (Wald and Brown, 1952 ; Red Fern and Strangeways, 1947 ; Mason and Ellison, 1935). For example, the process of keratinisation has been found to be associated with the disappearance of −SH group and a rapid increase in the concentration of −SS group (Pearse, 1954). Presumably, vitamin A prevents the conversion of −SH to −SS group, which is essentially a process of oxidation. The fibrous protein, keratin is characterised by its high content of the basic amino acids arginine, lysine and histidine and of the sulphur containing amino-acid cystine. Non-keratinised skin, on the other hand, has a high concentration of methionine. It would thus appear that though keratinisation is regarded as a manifestation of vitamin A deficiency, the underlying mechanism, however, is a disburbance in the metabolism of amino acids. Can vitamin A act effectively as an antikeratinising factor if the supply of methionine is severely limited ? That methionine plays a very important part in the maintenance of −SH group in some vital tissues like the liver and lens has been conclusively proved (Neuberger and Leaf, 1947 ; Bagchi, 1957, 1958). Protein malnutrition, has been found to affect the enzyme concentration in tissues. Heard (1957) observed in low protein feeding, a change in the pattern of enzyme concentration and distribution. Intracellular enzymes like glutathione reductase, which play a vital part in maintaining glutathione (Pirie and Van Heyningen, 1956) and other −SH group and thereby maintaining the functional part of many important intracellular enzymes, are all dependent on the protein supply.

Considering the available evidence cited above, one might suggest that the proper utilisation of vitamin A for the cellular metabolism would be adversely affected, if there is an associated protein malnutrition. In other words, supplying of vitamin A to the tissues, which are at the same time severely depleted of protein, will not help much in the reversal of the tissue changes.

The good clinical response with vitamin A and protein is also associated with increase of serum protein, albumin and correction of A:G ratio. Moreover, as discussed before, pigmentation and capillary proliferation and engorgement have been observed to be intermediate stages, both in the development and regression of Bitot's spot. Both these features, while having no recognised correlation with vitamin A deficiency, are closely linked with protein malnutrition. Circumcorneal and corneal infiltration with capillaries (Hock *et al*, 1945) and pigmentation or dyspigmentation of different parts of the body have been observed in recent years to be manifestations of protein malnutrition and amino-acid deficiency (Brock and Antret, 1952).

There is another significant observation regarding the aetiology of Bitot's spot which deserves mention. While conducting nutritional surveys in rural and urban areas, especially in the slums of Calcutta, it was surprising to observe the comparative rarity of Bitot's spot in rural areas as compared to its incidence amongst the urban population, the vitamin A and carotene intake of the diet in the two groups being almost comparable. Could it be due to some environmental factors present in the slums of the urban areas which aggravated or precipitated the condition ? Almost all cases of Bitot's spot in the present investigation came from a very low socio-economic group in which the living condition was extremely poor. Each family lived in a small room with almost no ventilation and the cooking was done in front of the room, usually in a small verandah. During lighting of ovens in the morning and afternoon,

the rooms remained stuffed with smoke for a very long time. In fact, during at least four hours a day the entire area of the slum was covered by a thick smoke emanating from a large number of rooms comprising the area. This, certainly, is a source of irritation for the eyes. This factor, it seems, would be of significance, if the site of Bitot's spot is considered. The spot appears only in the triangular patch, coinciding with the triangular palpebral opening or in other words, the portion of scleral conjunctiva which is most exposed to the atmosphere. Naturally, it is quite pertinent to assume that this chronic source of irritation might be a contributory factor. In the rural area, the houses are largely scattered and even the poorest families have a separate hutment for cooking purposes and as such this source of irritation is not there.

Conclusion

The present investigation throws considerable light on the mechanism of genesis of Bitot's spot and unveils the composite factors that are operative in the development of the triangular foamy patches. The present conception of a vitamin A deficient diet is no longer tenable. Simple vitamin A deficiency in an otherwise healthy body may not give rise to Bitot's spot. Protein malnutrition, apart from interfering with the absorption, storage and transport of vitamin A probably interferes with its utilisation in tissues. Vitamin A deficiency superimposed on protein malnutrition might be responsible for Bitot's spot. The hypothesis is strongly corroborated by clinical response obtained after a combined therapy with vitamin A and protein whereas treatment with vitamin A alone even in large doses fails to produce effect in most of the cases. Moreover, some external factor like exposure to dust and smoke producing chronic irritation of the exposed part of the conjunctiva may also play some part.

Acknowledgments

Grateful thanks are due to Dr. A. R. Sundararajan, Professor of Biochemistry & Nutrition and Dr. N. Jungalwalla, Director of this Institute for the keen interest in this work. Statistical analysis was done by Sri S. C. Bhowmick of the Section of Statistics of this Institute for which the authors are deeply indebted to him.

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ANAEMIA IN CIRRHOSIS OF THE LIVER

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Introduction

Hepatic cirrhosis was described as a clinico-pathological condition as early as 1826 by Laennec. The occurrence of anaemia in cirrhosis was recorded as late as 1889 by Ham. Gram (1853), quoted by Meulengracht (1922), described macrocytosis by measurement of the cell diameter. Following these earlier reports, cases of anaemia based mostly on single case or a small number of cases were recorded by Cabot (1897), Haan (1898), Limbeck (1901), Perrin (1908), Emerson (1906),

Telley (1908), Naegeli (1912), Turk (1912), Babonniex and Tixier (1913), Roth (1914) and Eppinger (1920). The role of liver in the haemopoiesis was overlooked till the days of Minot and Murphy (1926). They instituted the treatment of pernicious anaemia with raw liver based on the experimental results of Whipple *et al* (1920) and of Whipple and Robschiet-Robbins (1925). Since there is extensive damage to the liver parenchyma in cirrhosis, a dyshaemopoietic factor in the pathogenesis of anaemia could not be overlooked. However, there was absence of agreement amongst the various workers regarding the types of anaemia met with in cirrhosis of the liver.

Watson (1939) suggested the presence of a haemolytic agent in some cases of cirrhosis. Davidson and Fullerton (1938) showed increased fragility in one case of cirrhosis. Singer and Dameshek (1941) recorded haemolytic anaemia in subacute hepatitis. Coleman (1948) reported two cases of cirrhosis with haemolytic anaemia and more recently Jandl (1955) observed the presence of haemolytic element in some of his cases.

Huang and Wang (1949) reported normocytic anaemia in most of their cases of cirrhosis of the liver in China. Wintrobe (1936) from America also found a similar morphological type of anaemia in some of his cases.

A large number of reports from different countries has accumulated in support of the occurrence of a macrocytic anaemia in cirrhosis—Wintrobe and Shumaker (1933), Wintrobe (1936), Rosenberg and Walters (1936) and Berman *et al* (1949) from U.S.A. ; Schalm (1937), Larsen (1948) Brarier (1948) and Mario (1951) from the Scandinavian countries ; Gamna (1926 and 1933) and Perelli (1928) from Italy ; Schulten and Malamos (1932), Fellinger and Klima (1933) and Malamos (1935) from Germiany ; Benhamou and Nouchi (1937) from Algeria ; Davidson and Fullerton (1938) from the United Kingdom and from Ceylon by Fernando *et al* (1948). These reports indicate that there may be some other factors besides liver injury to account for the anaemia and that the same may vary from one country to another.

Cirrhosis of liver is fairly common in India. But the problem of anaemia in cirrhosis has not been seriously investigated in this country.

The present work was undertaken to investigate the type and pathogenesis of anaemia in cirrhosis.

Materials and Methods

Detailed haematological studies have been made on 53 cases of cirrhosis admitted into the medical wards of the Nilratan Sircar Medical College Hospital, Calcutta and S. S. K. M. Hospital's surgical wards. In the selection of the cases patients receiving any haematinic treatment were excluded from the series. The patients were investigated immediately after admission or within a few days before any active treatment was started. These cases were diagnosed clinically and subsequently confirmed by a battery of liver function tests including electrophoresis of serum and also by needle or surgical biopsy of the liver (Aikat and Sengupta, 1958).

Complete cytological examination of the blood, including the thrombocyte count, the haematocrit and the mean corpuscular values were done by the methods of Dacie (1950).

Serum total direct bilirubin and total indirect bilirubin (Malloy and Evelyn, 1939) and direct and indirect quotient (Gray and Whidborne, 1946a and 1946b) were also determined at the same time.

The sternal bone marrow was studied in detail in 32 cases.

The secretory function of gastric mucosa was studied by fractional gastric analysis (with 7 per cent alcohol test meal) in 35 cases.

Faecal urobilinogen was estimated (MacLagan, 1946) in 45 cases.

Apart from these, routine laboratory investigations were done in each case to exclude any source of infection or infestation as cause of anaemia. These included.

(1) Blood : (a) for malarial parasities and for microfilaria, (b) complement fixation test for kala-azar, (c) Wassermann reaction, Kahn and V.D.R.L.

(2) Complete physical, chemical and microscopical examination of urine.

(3) Examination of stool specially for ova, protozoa and cysts. Helminthic ova were searched for by the salt flotation method.

Results

(A) Haemoglobin red blood cells M.C.V., M.C.H.C., M.C.H. The different haematological data have been summarised in Table 1.

Table 1—Showing Different Haematological Data from 53 Cases of Cirrhosis of the Liver

Different data	Range	Mean	Standard error
Haemoglobin (g. per cent)	2·38— 12·80	9·49	0·21
Red blood cells (million per cmm.)	1·25— 4·20	3·31	0·10
Haematocrit (per cent) ..	10·0 — 42·0	31·47	1·04
M.C.V. (*cu*) ...	70·0 —125·0	94·34	1·44
M.C.H.C. (per cent) ..	23·8 — 40·0	29·97	0·52
M.C.H. (*uug*) ...	21·3 — 37·2	28·72	0·47
Reticulocyte (per cent) ...	1·0 — 16·00	1·81	0·14

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Orthrochromia was observed in 38 (79 per cent) cases and hypochromia in 15 (29 per cent) cases. Thus orthochromia was more than twice as frequent as hypochromia.

The cases have been classified into different haematological groups as presented in 'Scatter' distribution with reference to mean corpuscular volume (M.C.V.) and mean corpuscular haemoglobin concentration (M.C.H.C.) (Fig. 1).

From the 'Scattergram', it was evident that although most of these cases were in the normocytic group, i.e., with M.C.V. between 80-100 cubic microns, a 'tendency' to macrocytosis was evident from the fact that 13 (33 per cent) cases out of 38 with normocytosis were seen to have a M.C.V. above 94 while only 25 cases fell within the rather wider range of 80-94 cubic microns.

The cases with macrocytosis were observed to have less severe degree of anaemia than those with the other morphological types (Table 2).

(B) Reticulocyte—the range of reticulocyte count was observed to vary from 1 to 16 per cent. The reticulocyte count was not related either to the type or degree of anaemia.

(C) Morphology of peripheral blood smear—(i) Anisocytosis and poikilocytosis of varying degree were observed in some cases usually associated with severe degree of anaemia. (ii) Target cells—these cells were found in 30 (56·6 per cent) cases (Fig. 2, *vide* Plate).

All cases showing hypochromia were associated with presence of fair number of target cells in their peripheral blood. The presence of hyperbilirubinaemia appeared to have some relation with presence of target cells in circulation (Table 3). Thus, it appears that appearance of target cells in the peripheral may be influenced by (1) hypochromicity and (2) hyperbilirubinaemia or both.

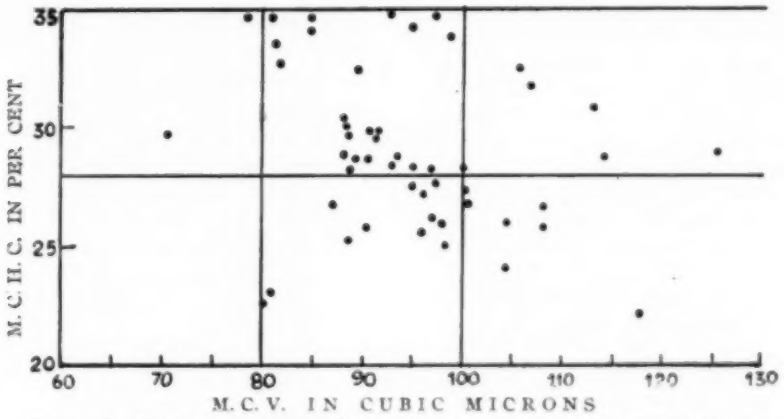

FIG. 1—'SCATTER' DISTRIBUTION OF 53 CASES OF CIRRHOSIS OF THE LIVER, CLASSIFIED INTO HAEMATOLOGICAL GROUPS WITH REFERENCE TO MEAN CORPUSCULAR VOLUME (M.C.V.) IN CUBIC MICRONS AND MEAN CORPUSCULAR HAEMOGLOBIN CONCENTRATION (M.C.H.C.) IN PER CENT.

TABLE 2—SHOWING RELATION OF HAEMATOLOGICAL GROUPS OF ANAEMIA WITH DEGREE OF ANAEMIA IN CIRRHOSIS OF THE LIVER

Haematological groups of anaemia	Total No. of cases	Number of cases with packed cell volume in per cent						
		0-15	15-20	20-25	25-30	30-35	35-40	40-45
Macrocytic orthochromic ..	7	—	2	—	2	—	3	—
Macrocytic hypochromic ...	6	—	—	—	—	2*	1	3
Normocytic orthochromic ...	29	—	—	4	9	8	7	1
Normocytic hypochromic ...	9	1	2	—	2	1	—	3
Microcytic orthochromic ...	2	—	—	1	—	1	—	—

TABLE 3—SHOWING RELATION OF INDIRECT BILIRUBIN IN MG. PER CENT WITH INCIDENCE OF TARGET CELLS IN CIRRHOSIS OF THE LIVER

Bilirubin in mg. per cent	Total No. of cases	Incidence of target cells	
		No. of cases	Per cent
Less than 1·0 ...	29	14	48
More than 1·0 ...	22	16	72

(D) Leucocytes: (i) Total count of leucocytes—Normal range of white cells were taken to be 5000 to 10,000 per cmm. Any count above 10,000 was taken to be leucocytosis and below 4000 as leucopenia. Leucopenia was frequently associated with splenic enlargement and the single case with leucocytosis had no enlargement of spleen. (ii) Differential count of leucocytes—The normal range of absolute figures for neutrophils, lymphocytes and eosinophils are 3000-5800 per cmm., 1500-4000 per cmm. and 150-400 per cmm. respectively (Whitby and Britton, 1953). Neutropenia, i.e., absolute figure below 3000 was observed in a very large number of cases (30 out of 43). Lymphopenia (below 1500) was seen in almost all the cases.

(E) Thrombocytes: Thrombocytopenia was observed in 23 out of 38 cases (60 per cent).

(F) Fractional gastric analysis: The criteria of analysis were based on observations of Napier and Das Gupta (1935) and Napier *et al* (1938). The results have been summarised (Table 4). Since gastric acidity could be one of the factors concerned with iron absorption, hypochlorhydria or achlorhydria might be related to the cause of hypochromic anaemia. Hypochlorhydria was the most frequent association, while achlorhydria was seen only in 2 cases.

TABLE 4—SHOWING RESULTS OF FRACTIONAL GASTRIC ANALYSIS OF 35 CASES OF CIRRHOSIS OF THE LIVER

Haematological groups	Hyperchlorhydria	Isochlorhydria			Hypochlorhydria	Achlorhydria
		High	Medium	Low		
Macrocytic orthochromic ...	—	—	—	—	4	1
Macrocytic hypochromic ...	—	—	1	—	4	1
Normocytic orthochromic ...	—	—	4	5	8	—
Normocytic hypochromic ...	—	—	—	—	5	—
Microcytic orthochromic ..	—	—	1	1	—	—
Total ...	—	—	6	6	21	2

(G) Bilirubin: (i) Total direct and indirect bilirubin—Large number of cases in different haematological groups of anaemia had total direct bilirubin between 0·2 and 1·0 mg. per cent whereas 29 cases of normocytic groups were within the above range. There was no correlation with direct and indirect bilirubin content either with the degree of anaemia or with the different morphological groups. Increased reticulocyte count was, however, observed in 21 out of 28 cases (75 per cent) within normal range of total indirect bilirubin.

(ii) Direct-indirect-quotient (DIQ)—High DIQ, i.e., above 71 was observed to be frequently associated with macrocytic group of anaemia (Table 5). DIQ was, however, not related to the degree of anaemia.

TABLE 5—SHOWING RELATION OF HAEMATOLOGICAL GROUPS OF ANAEMIA WITH DIRECT-INDIRECT-QUOTIENT IN CIRRHOSIS OF THE LIVER

Haematological groups of anaemia	Total No. of cases	No. of cases with direct-indirect-quotient			
		40-50	51-60	61-70	Above 71
Macrocytic orthochromic ...	6	—	1	3	2
Macrocytic hypochromic ...	5	—	—	2	3
Normocytic orthochromic ..	29	9	8	8	4
Normocytic hypochromic ...	9	4	4	1	—
Microcytic orthochromic ...	2	2	—	—	—

(H) Faecal urobilinogen: The range of faecal urobilinogen in 17 out of 45 cases varied between 238 mg. and 336 mg. per 100 g. of faeces (Fig. 3). There was no correlation with the degree of anaemia and faecal urobilinogen content. Reticulocyte per cent varied from 1·2 to 4·0 in the above 17 cases.

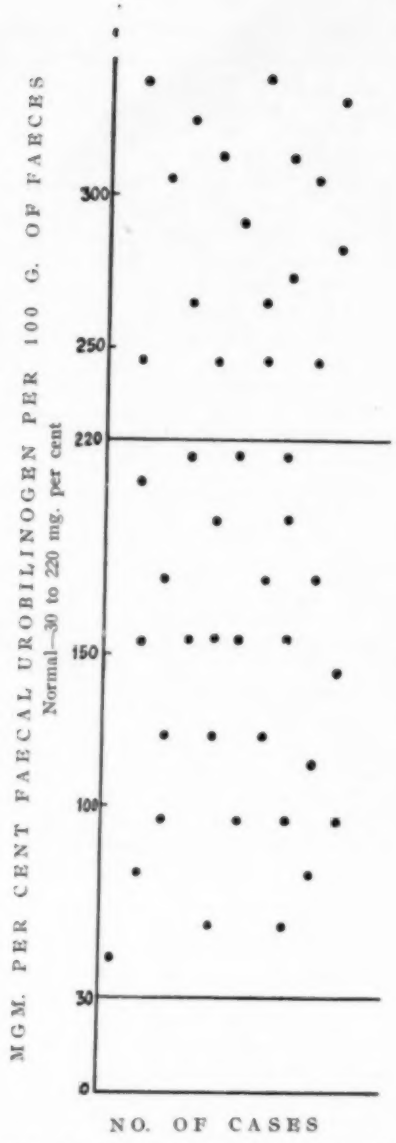

FIG. 3.—'SCATTER' DISTRIBUTION OF FAECAL UROBILINOGEN IN MG. PER 100 G. OF FAECES IN 45 CASES.

(I) Bone marrow: Reversal of ME ratio with increased or normal cellularity of the marow.

Normoblastic hyperplasia of the marrow with macronormoblasts (Fig. 4, *vide* Plate) was present in 50 per cent of macrocytic anaemia only (where the sternal marrow was studied) and independent of bleeding. Increased percentage of basophilic normoblasts were present in 11 cases whereas another 16 cases showed increased percentage of late normoblasts (Table 6).

TABLE 6—SHOWING DIFFERENTIAL COUNT OF MARROW CELLS IN 32 CASES OF CIRRHOSIS OF THE LIVER

Type of marrow cells	Range	Mean	Standard error
Proerythroblast ...	0·2— 3·2	1·04	0·13
Normoblast A ...	1·2— 6·6	3·14	0·38
Normoblast B ...	2·4—30·0	11·81	0·98
Normoblast C ...	8·0—36·8	22·59	1·46
Myeloblast ...	0·0— 1·2	0·62	0·11
Promyelocyte ...	0·0— 5·2	1·70	0·23
Neutrophilic myelocyte ...	3·0—20·6	13·57	0·73
Eosinophilic myelocyte ...	0·1— 4·0	1·99	0·18
Basophilic myelocyte ...	0·0— 0·2		
Neutrophilic metamyelocyte ...	7·2—24·0	15·28	0·73
Eosinophilic metamyelocyte ...	0·0— 7·8	2·01	0·33
Basophilic metamyelocyte	0·0— 0·2		
Polymorphonuclear neutrophil	6·0—27·6	18·14	0·88
Polymorphonuclear eosinophil	0·4— 9·8	2·52	0·36
Polymorphonuclear basophil	0·0— 0·5		
Lymphocyte	0·4— 9·6	5·24	0·48
Plasma cell	0·0— 3·0	1·25	0·16
Monocyte ...	0·0— 3·0	1·33	0·17

In 3 cases of macrocytic anaemia, giant metamyelocytes (Fig. 5, *vide* Plate) were observed. Any other significant changes in the granulocyte series were not observed.

Increased plasma cells were found to be related in a fair number of cases to hyperglobulinaemia.

Megakaryocytes were normal in frequency and in morphology.

DISCUSSION

The concept of dyshaemopoietic anaemia in cirrhosis of the liver was introduced by Robscheit-Robbins and Whipple (1933) who attributed the low haemoglobin production to severe liver injury (Wintrobe and Shumaker, 1933; Wintrobe, 1936). To this was added the factor of gastric secretory deficiency (Wintrobe and Shumaker, *loc. cit.*; Davidson and Fullerton, 1938). Since evidence of complete gastric secretory deficiency was not obtained in any case in the present series, absence of intrinsic factor seemed to be untenable.

The dyshaemopoietic anaemia appeared to have received support from the observation of megaloblastic bone marrow in some cases by Isaacs (1935 and 1937) and, recently, by de Castro (1946) Jarrold and Vilter (1949), Leitner et al (1949), Movitt (1949, 1950). In the present investigation,

on the other hand, marrow changes were characterised predominantly by normoblastic hyperplasia with macronormoblasts observed only in a limited number of cases. The cellularity, in general, was normal or increased (Zanaty, 1937 ; Isreals, 1941 ; Jones, 1943 ; Huang and Wang, 1949 ; Berman *et al*, 1949). The remissions with crude liver extract observed by various authors (Bernheim, 1924 ; Goodhart, 1932 ; Goldhamer *et al*, 1934 ; Movitt, 1950) indicated the probable role of deficiency of liver principles in the pathogenesis. A large number of workers, on the other hand, have reported absence of any significant response to even recent haematinic drugs (Brarier, 1948 ; Jarrold and Vilter, 1949 ; Bateman *et al*, 1949 ; Mario, 1951). Owren (1951) believed that the liver takes a very important part in the final transformation of vitamin B_{12} and folic acid, into the most active 'erythrocyte maturation factor'. The failure to restore the blood picture in cirrhosis using modern haematinics could be explained on deranged protein synthesis by the damaged liver. A recent observation of Larsen (1948) suggested that deficiency of nicotinic acid might play a part in the process whereas Brarier (1948) postulated that it depended on some complex disturbance of metabolism and of nutrition.

Remissions in anaemia were considered to be a further point in favour of dyshaemopoietic type of anaemia similar to PA (Gram, 1853, quoted by Meulengracht, 1922 ; Stewart, 1917 ; Van Duyn, 1933 ; Rosenberg and Walters, 1936). Huang and Wang (1949), however, suggested that increased incidence of megaloblastic anaemia in the continent and its frequent association with cirrhosis of liver might explain the above discrepancy. They found macrocytosis in a small percentage of their cases (2 out of 32) along with macronormoblastic marrow.

Rosenberg and Walters (1936), based on the findings of Snell (1935), suggested hypo-albuminaemia as a physicochemical factor in the genesis of macrocytosis. Since macrocytosis was seen in about one fourth of the cases showing hypoalbuminaemia, colloidal osmotic factor alone cannot explain the cause of the increased size of the cell in all such cases.

In the present investigation, the direct-indirect-quotient (DIQ) of bilirubin appeared to be related to macrocytosis (Table 5). This association of macrocytosis with high DIQ in the present series offers suggestive evidence that macrocytosis occurred when the balance in the two kinds of bilirubins altered.

Although the tendency to develop a macrocytic anaemia was noted by Wright (1935), Isaacs (1937), the transition from a microcytic or normocytic anaemia to macrocytic type was reported by Van Duyn (1933), Goodhart (1932) and Rosenberg and Walters (1936) as an accompaniment of increasing liver damage. The present investigation confirms the above observation on 'tendency' to macrocytosis as 13 out of the 38 cases of the normocytic group had their mean corpuscular volume between 94-100 cubic microns (Fig. 1.).

Rosenberg and Walters (1936) suggested that reticulocytes in large number might be responsible for macrocytosis. Only one case in the present series had a very high reticulocyte count (16 per cent) which would account for the macrocytosis in this single case.

Association of hypochromia in 15 cases would point to deficiency of iron. There was absence of hydrochloric acid in 2 and hypochlorhydria in another 21 cases. This hypochlorhydria and achlorhydria in 23 out of 35 cases could perhaps to some extent explain the deficiency in patients who were already on the 'border-line diet'.

Giant metamyelocytes were observed in marrow smear in 3 of 8 cases of the macrocytic group. These cells are usually associated with megaloblastic erythropoiesis (Henning, 1935). Their association with dimorphic anaemia was reported by Trowell (1943) and Chatterjea (1949). Absence of megaloblasts and presence of normoblastic marrow may be due to combined deficiency of iron and liver principle (Trowell, 1943 ; Chatterjea, 1949) as in some cases of nutritional macrocytic anaemia.

Leucopenia and thromocytopenia, which are usually associated with dyshaemopoietic anaemia, were uniformly present in a large majority of the cases.

The above evidences would suggest that the nutritional factors may play a very important role in the genesis of anaemia in cirrhosis of the liver.

History of haemorrhage was obtained in only 4 cases one week or more before admission in the hospital. Routine examination of stool for occult blood was positive in three other cases. Anaemia attributable to a predominant haemorrhagic episode could operate only in a limited number of cases in this series. Although hypochromia and macronormoblastic bone marrow could be explained on the basis of chronic haemorrhage, this could not be substantiated on the available evidences.

Anaemia with normoblastic marrow and leucopenia were observed along with the enlarged spleen in 23 out of 53 cases of cirrhosis of liver. These findings have been explained on the basis of hypersplenism (Doan and Wright, 1946 ; Dameshek and Estren, 1947). 'Adrenaline test' done in 12 cases, however, proved essentially negative in all the instances (Sengupta and Aikat, 1959a). Some improvement in the degree of

anaemia, observed in some of our cases with enlarged spleen following splenectomy, would suggest the possibility of hormonal inhibition of bone marrow in those cases (Dameshek and Estren, 1947).

Hypervolaemia has been recognised as one of the causes of anaemia. In the present series hypervolaemia was observed in 6 out of 45 cases (Sengupta and Aikat, 1959b). These cases, showed hyperplastic changes in their bone marrow. Bateman *et al* (*loc. cit.*), however, did not observe any marrow changes in cases of cirrhosis of liver with hypervolaemic anaemia. Thus hypervolaemia may be a contributory factor in enhancing the degree of anaemia in some cases of cirrhosis of liver.

In 17 out of 45 cases, there was high excretion of faecal urobilinogen. All these cases showed minor degree of reticulocytosis and hyperplastic marrow. These are very suggestive evidence of the interplay of a chronic haemolytic process. Acute episodes of haemolysis in cirrhosis of liver have been reported by van den Bergh (1930), Coleman (1948) and in hepatitis by Hyman and Southworth (1951). Jandl (1955) demonstrated evidence of haemolysis in some of his cases. Mechanism of haemolysis may be due to abnormality in the red cells or abnormal haemolytic mechanism in the spleen. Jandl (*loc. cit.*) did not observe any diminution in the life span of tagged red cells. He suggested an abnormal splenic haemolytic mechanism as the causes of increased destruction of red blood cells. In 8 out of these 17 cases with increased faecal urobilinogen there was no splenic enlargement. A further search for the site of red blood cell destruction apart from the spleen, with chromium tagged red cells may serve to unravel newer facts on the complex problem.

From the present study, it would be evident that the cause of anaemia in hepatic cirrhosis could not be attributed to a single factor. Dyshaemopoiesis, haemorrhage, hypersplenism, hypervolaemia and haemolysis may operate alone or in combination in causing anaemia in cirrhosis of the liver.

Summary and Conclusion

In a study of 53 cases of cirrhosis of the liver the types of anaemia were found to be macrocytic in 13 (24·5 per cent), microcytic in 2 (3·8 per cent) and normocytic in 38 (71·7 per cent) About one third (13 cases) of the normocytic group showed a 'tendency' to macrocytosis because these cases had their mean corpuscular volume between 94—100 microns.

Macrocytosis in these 13 cases was found to be related to the direct-indirect-quotient of the plasma. Direct-indirect-quotient indicates the relative amounts of direct and indirect bilirubin present in the plasma.

Microcytic anaemia was found only in those cases with history of haemorrhage.

Association of hypochromia in 15 cases might be to some extent due to hypochlorhydria or achlorhydria.

From the study of faecal urobilinogen excretion per 100 g. of faeces there was evidence of a little high excretion rate in 17 cases. Thus in a fair percentage of cases, haemolysis was a factor contributing to the anaemia. It appears that no single factor can be held responsible for the anaemia in cirrhosis. Hypervolaemia, haemorrhage, dyshaemopoiesis and haemolysis may operate singly or in combination.

Acknowledgment

We are grateful to Major General A. K. Gupta, Director, Institute of Post-Graduate Medical Education & Research, for allowing us to publish the article. Our thanks are due to Lt. Col. N. C. Chatterjee, formerly Surgeon Superintendent, S. S. K. M. Hospital, Dr. A. K. Dutta Gupta, Principal, N. R. S. Medical College Hospital, and Dr. A. K. Basu, Director-Professor, Department of Surgery, Institute of Post-Graduate Medical Education & Research, Calcutta.

Thanks are also due to Dr. J. B. Chatterjea, Professor of Haematology, School of Tropical Medicine, for his helpful criticism.

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FATAL COMPLICATION WITH PREDNISOLONE THERAPY IN NEPHROTIC SYNDROME—WITH A CASE REPORT

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Of recent advance in the management of nephrotic syndrome, has been the use of steroids both as short-term therapy or for maintenance over prolonged periods. During the last few years the place of these hormones has been firmly established in the abolition of oedema and proteinuria and with the introduction of prednisolone the risks entailed in such therapy were considerably reduced. Trials with short courses of prednisolone in patients suffering from nephrotic syndrome have already been report-

ed (Arneil, 1956 ; Mathur, Prasad and Srivastava, 1959) and though in our series we had not come across any complication, we had been impressed by the likelihood of such an occurrence and had therefore resorted to only small dosage schemes extending for a period of only one to two weeks. The occurrence of one fatal complication during treatment with prednisolone, which is being reported here, prompts us to make a fresh plea for careful use of such potent hormones.

Case Report

A 30 year old male was hospitalised on 2-7-1958 with 5 months' history of generalised oedema. The onset was insidious and oedema first appeared over the lower extremities. On examination he was slightly pale, had moderate oedema all over, body weight was 92 lb., B.P. 118/76 mm. of Hg, urine contained 0·1 to 0·25 g. per cent albumin, a few hyaline and granular casts. Blood urea and nonprotein nitrogen were 30 mg. and 24 mg. per cent respectively, serum cholesterol was 310 mg. per cent, total plasma protein 5·3 g. per cent with 3 g. per cent of albumin and 2·3 g. per cent of globulin. Kidney functions were well maintained. Other systems were normal and radiograph of the lungs did not reveal any pathology. Congo red test was negative. Needle renal biopsy was performed on 10-7-1958 and revealed moderate amyloidosis of the kidney (Fig. 1, *vide* Plate). Liver biopsy did not show any hepatic disease. The patient was put on prednisolone 15 mg. per day for 10 days and, in view of renal amyloidosis as the histological diagnosis, showed unexpected symptomatic relief with marked diuresis, diminution of proteinuria and abolition of oedema. When oedema-free his body weight had come down to 78 lb., but he was still passing traces of albumin in the urine. He was discharged from the hospital on 5-8-1958 and was followed up in an oedema-free state for one more month and was then lost trace of.

He next sought admission on 22-5-1959 with recurrence of oedema, this time of about 6 months' duration (thus he had remained oedema-free for about 3½ months) and also complained of irregular fever and cough. Examination revealed moderate oedema, body weight 90 lb., B.P. 136/86 mm. of Hg, gross proteinuria with presence of casts. Blood urea was 42 mg. per cent while the cholesterol and protein content of blood were unaltered from the first examination. Renal functions were still normal. Skiagram chest this time revealed infiltration in the right subclavicular zone, sputum however was negative for acid-fast bacilli. He was again put on prednisolone therapy, 15 mg. per day from 3-6-1959 along with streptomycin and isonicotinic acid hydrazide. During the first ten days of prednisolone therapy, he showed a good response with marked diminution of oedema, and general sense of well being. But, on 13-6-1959 he developed oliguria, oedema recurred and by evening he developed cellulitis over the left cheek with temperature of 99·2°F. Prednisolone was stopped and he was put on 2 mega units of penicillin G sodium daily by intramuscular route, but the cellulitis spread over the whole of the face and neck, the temperature ranged between 100° and 101°F. The patient became markedly toxaemic and died on 15-6-1959, i.e., within 48 hours of recognition of cellulitis.

Comments

The complications or side-effects met with in the use of ACTH, cortisone or other newer corticosteroids are mostly due to the profound biochemical changes which are induced. In addition to the known hazards, there are certain unusual risks in the treatment of patients with nephrotic syndrome. The most dreaded of these is the spread of infection. Symptoms and signs of infection are often concealed by the antipyretic and anti- inflammatory effects of these hormones, and clinical flare-up occurs frequently when thrapy is stopped. The leucocytosis that develops regularly with steroid treatment, in the absence of clinical signs, cannot be relied upon as evidence of active infection and therefore adds to difficulty in early recognition of this complication. Ross and Ross (1957) have reported two deaths from spreading cellulitis during treatment with prednisolone in the nephrotic syndrome. One of their patients had prednisolone 20 mg. twice a day for 6 days and then 10 mg. twice daily. He developed cellulitis on the 14th day of treatment and died within 48 hours. The other case was put on longer dosage schedule with 20 mg. b.d. for one month and was then put on maintenance of 10 mg. b.d. and after 2 weeks on this maintenance dose developed spreading cellulitis and died on the 4th day.

The case reported here was suffering from renal amyloid disease and nephrotic syndrome, and at the first observation in 1958 a therapeutic trial with prednisolone was made only to see if such cases showed any response. We were rather surprised to see a good response, and though we were not able to follow up this patient, we had considered a clinical relapse almost inevitable in view of the renal pathology and protein leak in urine which had persisted inspite of treatment (Mathur *et al, loc. cit.*). It was therefore with this background that we decided to give another trial to prednisolone when he came for observation for the second time in May, 1959. Development of cellulitis was sudden and in view of the fact that the patient was neither grossly oedematous nor seriously ill we feel that occurrence of cellulitis would not normally have been considered a serious risk. It is concluded that the fulminating nature of the infection was due to the use of prednisolone and the occurrence of this fatal complication has impressed upon us the necessity of early recognition of infection and vigorous treatment. Since early recognition is difficult, we are

now of opinion that prophylactic penicillin therapy may materially lower this risk of infection during treatment with corticosteroids.

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ERYTHROCYTE SEDIMENTATION RATE IN COMA CAUSED BY INFECTIVE HEPATITIS

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INTRODUCTION

While the vast majority of patients suffering from infective hepatitis proceeds to uneventful recovery, a small percentage with its fulminant variety follows a downhill course into coma. Coma caused by infective hepatitis is the most serious and dreaded episode in the natural history of the disease, presenting a grave threat to the patient's life owing to its high mortality rate, and thus throwing an enormous challenge to the physician.

The association of mental disturbances with hepatic disease has been known since the days of Galen, but even today the underlying mechanism is far from clear—being either a breakdown in the detoxicating function of the liver or liberation of a toxic by-product of its disordered metabolism.

The present work was undertaken to study the changes in the erythrocyte sedimentation rate, specially with regard to its prognostic value, in coma caused by infectivev hepatitis. Literature on this subject is meagre, although various authors have studied its changes in infective hepatitis uncomplicated with coma.

MATERIAL AND METHODS

During the period January 1st, 1956 to June 30th, 1959 fifteen cases of coma caused by infective hepatitis were treated in the Medical Wards of the J. A. Group of Hospitals, Gwalior, M. P. Westergren's method of estimating the erythrocyte sedimentation rate was followed. The present paper is based on a study of these 15 cases.

The findings of the cases studied and the statistical analysis of the results are tabulated (Tables 1, 2 and 3).

OBSERVATIONS AND DISCUSSION

Gaur and Laha (Personal observation, 1958) established the normal erythrocyte sedimentation rate in this part of Madhya Pradesh as follows:

	Males	Females
Range	3—10 mm.	5—15 mm.
Mean	4·76	9·96
S. D.	±1·65	±2·10

The findings of the erythrocyte sedimentation rate in coma caused by infective hepatitis were studied in relation to the aforesaid normal findings.

The erythrocyte sedimentation rate was below 8 mm. during coma in the fifteen cases (all Hindus) of the present series, and in the majority (11 out of 15 cases) it was below 5 mm. In only two cases (No. 11 and 12) in whom the erythrocyte sedimentation rate could be estimated before the supervention of coma, it was 13 and 11 mm. respectively, but it came down to 4 and 3 mm. respectively as they (the cases) entered into coma. Repetition of the test in six (No. 3, 5, 8, 9, 12 and 13) out of the eleven fatal cases revealed persistence of the low sedimentation rate. In three cases (No. 1, 2 and 10) who came out of coma and survived, the erythrocyte sedimentation rate mounted high, being 14, 14 and 15 mm. respectively.

The aforesaid observations led the author to conclude that the erythrocyte sedimentation rate was low in infective hepatitis complicated with coma, persisted to be low in the fatal cases, started rising within six to eight hours before coma started clearing in those who survived, and the higher precoma rate fell down when the patients entered into coma. The fall of the higher precoma rate of sedimentation when coma supervened in the two cases (No. 11 and 12) was statistically highly significant (Table 2), and its rise in the three cases (No. 1, 2 and 10) who survived, was statistically significant (Table 3). It was evident that the erythrocyte sedimentation rate in infective hepatitis complicated with coma showed 'masked' reaction, i.e., it was low normal, although the clinical picture and condition of the liver led one to expect the contrary. 'Masked' low sedimentation rate is, in the experience of Wuhrman (1956), almost exclusively confined to those cases of dyspro-

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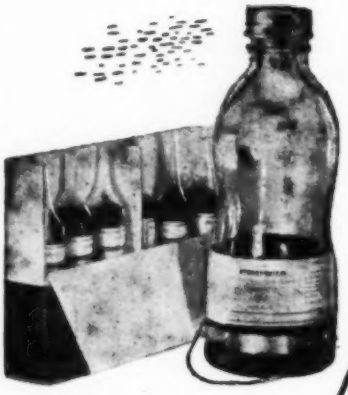

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TABLE 1—SHOWING FINDINGS OF THE CASES STUDIED

No.	Name	Age (years) sex	Duration of illness (days)	Duration of coma when investigations were done (hours)	INVESTIGATIONS			Termination after onset of coma
					E.S.R. in mm./1st hour (Westergren)	Thymol turbidity (units)	Serum bilirubin (mg. per cent)	
1	K.	25, M.	15	48	3	5·0	16·0	Survived after 102 hours
				96	14	12·0	30·0	
2	N.	19, M.	6	48	5	12·0	14·0	Survived after 80 hours
				72	14	16·0	20·0	
3	P.	18, M.	10	7	4	18·0	36·0	Expired after 22 hours
				16	3	21·0	32·0	
4	D.	40, M.	3	18	4	20·0	28·0	Expired after 22 hours
5	R.	32, M.	25	24	4	14·0	40·3	Expired after 62 hours
				48	3	13·4	38·0	
6	S.	26, M.	5	12	1	10·0	17·5	Expired after 24 hours
7	R.	65, F.	30	12	4	10·0	20·0	Expired after 24 hours
8	D. P.	35, M.	10	4	6	10·0	11·5	Expired after 34 hours
				28	6	12·0	18·0	
9	R. R.	25, M.	5	12	6	10·0	18·0	Expired after 42 hours
				36	6	18·0	22·0	
10	P. R.	25, M.	4	12	2	26·0	27·0	Survived after 108 hours
				36	1	32·0	32·0	
				60	2	30·0	28·0	
				84	2	14·0	27·0	
				102	15	9·0	22·0	
11	M. B.	30, F.	15	48*	13	12·0	12·5	Expired after 15 hours
				12	4	26·4	46·0	
12	R. S.	20, F.	15	24*	11	18·0	28·0	Expired after 70 hours
				6	3	20·0	41·0	
				18	2	24·0	45·0	
				42	2	24·0	46·0	
				66	2	26·0	49·0	
13	S.	32, M.	30	20	7	8·6	10·5	Expired after 50 hours
				44	3	9·0	15·0	
14	A. S.	18, M.	5	22	2	20·0	22·0	Expired after 26 hours
15	B. R.	45, M.	6	4	2	20·0	22·6	Expired after 17 hours

* Before coma supervened.

TABLE 2—SHOWING STATISTICAL ANALYSIS OF THE CHANGES IN THE ERYTHROCYTE SEDIMENTATION RATE IN CASES (NO. 11 AND 12) IN WHOM THE HIGHER PRECOMA RATE FELL DOWN WHEN COMA SUPERVENED

Mean	+9·0
S. D.	0
Calculated 't' ...	Infinitive
Tabulated 't' ...	1·960 at 95 per cent level

TABLE 3—SHOWING STATISTICAL ANALYSIS OF THE CHANGES IN THE ERYTHROCYTE SEDIMENTATION RATE IN CASES (NO. 1, 2 AND 10) WHO SURVIVED

Mean	+11·0
S. D.	± 2·0
Calculated 't'	8·305
Tabulated 't'	6·965 at 98 per cent level

teinaemia which are caused by cirrhosis or damage of the livevr, including hepatitis. According to Wuhrmann and Niggli, quoted by Wuhrmann (1956), it is found in those cases in which a decrease of the albumin fraction and increase of the γ-globulin fraction are accompanied by a reduction of the total protein concentration. The logarithm of the maximal sedimentation velocity is proportional to the concentration in the plasma of the specific substances causing agglutination. And this constituent is usually associated with the globulin fraction. The estimation of plasma proteins could not be done in the cases under review. Hence, no direct light could be thrown on the role played by plasma proteins in producing 'masked' sedimentation rate in those cases. But thymol turbidity test which was carried out in all the cases threw some light in this direction, as the

turbidity has been supposed to be due to increased amounts of γ-globulin concentration in the serum. It has aslo been mentioned that the precipitate in the thymol turbidity test is not only γ-globulin but also lipid containing β-globulin. From the present study it could be argued that increased amounts of γ-globulin and lipid containing β-globulin, which are held responsible for producing positive thymol turbidity test, resulted in the 'masked' sedimentation rate, but the facts that in two cases (No. 1 and 2) who came out of coma and survived, the thymol turbidity test showed stronger positive reaction albeit the erythrocyte sedimentation rate mounted up from the low figures and that its rise within such a short period as 24 to 48 hours could not be sufficient time interval for complete reversal of metabolic processes, were against this argument.

Miles (1945) found strong negative correlation between the height of erythrocyte sedimentation rate and serum bilirubin in infective hepatitis, but no such correlation could be observed in the present series of cases.

The total duration of illness, the duration of coma and the line of treatment adopted (which was uniform in all the cases) did not apparently produce any reflection in the 'masked' sedimentation rate in the cases under review.

The author was, therefore, posed with the question as to what could be the factor or factors responsible for the 'masked' low sedimentation rate in infective hepatitis when coma supervened, and its rise in those cases who survived. According to Houssay *et al* (1955) the most important factor is the concentration of fibrinogen to which the sedimentation is directly proportional ; globulin concentration is not so important, and serum albumin even less. When fibrinogen concentration diminishes, as in defibrinated blood and in some cases of shock (Houssay *et al, loc. cit.*) the erythrocyte sedimentation rate is very low. Fibrinogen is made in the liver but, unlike albumin, its production is not diminished in hepatitis. Only after a large portion of the liver is destroyed does the plasma fibrinogen concentration become lowered. It is possible that when infective hepatitis follows a fulminant course and leads to coma the production of fibrinogen is diminished, and thereby the erythrocyte sedimentation rate becomes low. But the very quick fall of the erythrocyte sedimentation rate (Cases 11 and 12) and a similar rise (Cases 1, 2 and 10) within a very short interval made it difficult to visualise such a rapid change in the production mechanism of fibrinogen by the damaged liver. But a state of defibrinated blood in the cases under review could not be completely ruled out in the absence of the estimation of the fibrinogen content of the plasma.

According to Bell *et al* (1957) this phenomenon of erythrocyte sedimentation rate is related to the fact that each red cell has a negative electric charge ; anything that increases the positive charge in the plasma favours rouleaux formation and the larger the rouleaux the more rapidly do they sediment. It has been established that variations in the sedimentation rate depend exclusively on plasmatic factors, and little or not at all on the erythrocytes. The author is of the opinion that when coma supervenes in infective hepatitis some biochemical changes are produced in the plasma due to which its positive charge decreases and thereby the rouleaux formation of erythrocytes is also diminished leading to 'masked' low erythrocyte sedimentation rate. In the non-fatal cases the biochemical changes in the plasma reverse, and thereby its positive charge increases with increased rouleaux formation and the consequent mounting up of the sedimentation rate. These changes in the non-fatal cases start manifesting before the coma shows clinical signs of clearing away. And therein lies the value of determination of erythrocyte sedimentation rate in infective hepatitis complicated with coma for prognostic appraisal in the non-fatal cases.

The author concluded that the fall of erythrocyte sedimentation rate in infective hepatitis may be the earliest indication of impending coma, 'masked' low sedimentation rate is an invariable feature of coma, it persists so in the fatal cases, and its rise during coma foretells a hopeful prognosis when there are no clinical evidences to indicate so. This test is a simple side-room investigation and could be of value in evaluating the course of this dreaded complication of infective hepatitis. But this should not indicate that this is the only one worthy of consideration. The author has not been able to throw direct light on the mechanism of the 'masked' low sedimentation in infective hepatitis complicated with coma and its mounting up in those cases in whom coma will clear away, but he has postulated a hypothesis which needs elaboration. Although more work is warranted on this subject, it is concluded that the serial determination of erythrocyte sedimentation rate in infective hepatitis complicated with coma offers potentially some prognostic clues with regard to the termination of coma.*

* Biphasic lowering of erythrocyte sedimentation rate, viz., within 10 days and again around 30 days of onset of infective hepatitis clearing up with recovery, is on record (Wood, P.—*Brit. M. J.*, **1** : 9, 1945; Robinson, P. —*Ibid*, **1** : 310, 1945). Hence, it may not be surprising to have similar results if a comatose case recovers.—Ed., *J. Indian M. A.*

Summary

A study has been made of the changes in the erythrocyte sedimentation rate in fifteen cases of infective hepatitis complicated with coma, especially from the point of view of its prognostic value.

A hypothesis has been postulated by the author regarding the mechanism responsible for the changes in the erythrocyte sedimentation rate in coma caused by infective hepatitis.

Acknowledgments

My thanks are due to Dr. A. S. Misra, Superintendent of the J. A. Group of Hospitals, Gwalior, for his valuable co-operation.

I am thankful to Dr. R. Phalke, Demonstrator in Medicine, G. R. Medical College, Gwalior, for his help in studying the cases.

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CLINICAL TRIAL WITH SULPHADIAZINE LIPID EMULSION

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Due to the everincreasing number of antibiotics and their widespread use in various common and uncommon bacterial infections, the sulpha compounds seem to have lost their old popularity and receded into the background.

Sulphonamides, however, are still very useful drugs and as such should regain their lost popularity. We have been extensively and effectively using the sulpha drugs in our outpatients and indoor cases of respiratory and gastrointestinal infections in infants and children. It is our personal observation that in many cases of these types of infections the combined use of sulpha drugs and antibiotics is more effective than when antibiotics are used alone and a smaller dose of antibiotics is necessary to suppress the acuteness of such infections. Moreover, when the acuteness of an infection subsides, the therapy is effectively continued with sulpha drug alone. With the use of the new long-acting sulphonamides we could now safely discard the old practice of repeated doses, which cause hardships to parents as well as to children, to maintain a satisfactory blood level of the drug. Hence a single-dose-a-day treatment with an oral preparation of a long-acting sulphonamide is welcome and advantageous without in any way prejudicing the effectiveness of the drug.

In this paper are reported the therapeutic effects of a long-acting sulphonamide, sulphadiazine lipid emulsion, in 29 cases of acute infections of infants and children.

Material and Method

Out of the 29 cases, admitted in the hospital the duration of illness before admission was 1-5 days in 27 cases and 7-12 days in the remaining 2. The ages varied from 1½ months to 12 years. The maximum number of cases (10) occurred in the age-group of 3-6 months. Table 1 shows the nature of infection and the number of cases under each head.

Table 1—Showing Nature of Infection and No. of Cases

Disease	No. of cases
Acute upper respiratory tract infection ...	10
Acute upper respiratory tract infection with diarrhoea	7
Acute gastro-enteritis	6
Acute dysentery	3
Bronchopneumonia	1
Acute cystitis	1
Whooping cough with fever	1

The children were given a preparation of sulphadiazine in highly emulsified pleasant-tasting vegetable oil base, in a concentration of 10 per cent suspension (5 c.c. equivalent to 0·5 g. of sulphadiazine). The drug was given orally each morning in a single dose of ½ g. children upto 2 years of age and 1 g. to children above that age. Alkaline mixture in appropriate doses was given thrice daily to all the cases. Sulphadiazine lipid emulsion was the sole antimicrobial agent used. The attending nurses and parents were emphatically asked to give extra water and fluids liberally by mouth. The urine output was regularly charted and routine urine examination was done daily. Each patient was examined daily to note the clinical effects of the drug as well as its side-reactions, if any.

R.B.C., and W.B.C. counts were done in all cases before and after the treatment. The duration of treatment varied from 1-9 days.

In cases with dysentery and gastro-enteritis appropriate dietetic measures were taken. No case with dehydration was selected for this study. No bacteriological study was done due to lack of facilities.

Results

19 cases were cured. Marked relief was obtained in 7 cases and moderate relief in 2 cases. The drug had to be omitted in 1 case.

Discussion

Sheth *et al* (1958) in a comparative study on the absorption of sulphadiazine as an aqueous suspension and a lipid emulsion in two groups of children, found that lipid emulsion was absorbed better and maintained a higher concentration for a longer time in blood as compared to sulphadiazine in aqueous suspension. Sulphadiazine in a single oral dose of 100 mg./lb. of body weight given as lipid emulsion maintained an average blood level of 10 mg./100 c.c. for 12 hours and above 6 mg./100 c.c. for 24 hours. This is fairly adequate to treat most of the common infections. When the same dose of aqueous suspension was given the average blood levels went below 5 mg./100 c.c. at the end of 8 hours. From our clinical trial, we feel that sulphadiazine lipid emulsion, even though given alone, possesses the efficacy to suppress various common respiratory and gastro-intestinal infections. The drug is ideally suited for the treatment of these common infections, on account of its cheapness, once a day dosage schedule and its quick effectiveness. Majority of the cases under review (22 cases) responded very well with only 2-4 days of treatment, especially when the child was brought very early. The drug was well-tolerated and readily accepted by the children.

In one case of bronchopneumonia in a child, aged 4 years, dyspnoea subsided and the temperature became normal on the 5th day of treatment with 1 g. of the drug daily. Cough, which persisted, required 2 more days of therapy, with ½ g. dose daily, to disappear. Another child, aged 6 years, had acute cystitis with severe dysuria and pyuria, the pus rendering the urine thick in consistency. The child was put on 1 g. lipid emulsion daily and on the 3rd day he became afebrile with marked diminution in pyuria and dysuria. Another child, 6 months old, suffering from whooping cough for 2 weeks and running moderate temperature for 2 days, was put on ½ g. emulsion daily. He became afebrile on the 5th day of treatment. Thus the drug suppressed the secondary infection quickly. It had no effect on the spasms of whooping cough.

The urine and blood examinations did not reveal any abnormality and were within normal range in all cases.

Side reactions occurred in 4 cases: (1) A 5-month old child, suffering from acute upper respiratory tract infection, developed anuria on the 5th day of treatment for 14 hours. This transient anuria was relieved by plenty of fluids and alkalies orally. The drug was subsequently stopped. (2) A child, 4 years old and suffering from acute gastro-enteritis, persistently vomited the drug on the first day and hence the drug had to be omitted and an alternative treatment instituted. (3) A child, 6 months old, suffering from acute upper respiratory infection, developed oliguria on the 2nd day of therapy but with plenty of fluids orally the urine output became normal in 24 hours. (4) A child, 6 months old and suffering from acute upper respiratory infection with moderate diarrhoea without dehydration, developed oliguria on the 2nd day of therapy, but she also recovered with plenty of oral fluids in 24 hours. Thus it is advisable, especially in young infants, to give plenty of fluids orally and chart the output of urine daily when this drug is used.

Summary

29 infants and children, mostly suffering from common respiratory and gastrointestinal infections, were given sulphadiazine lipid emulsion once daily by the oral route in a dose of ½-1 g. The results were excellent in 19 cases and satisfactory in 9 others. 4 cases developed side reactions, one persistently vomited the drug, two developed oliguria and one had transient anuria.

Acknowledgments

I thank Dr. R. P. Vyas, Civil Surgeon, Bhavnagar, for his kind permission to publish this paper.

I also thank Shri A. V. Mody of Unichem Laboratories, Bombay, for the supply of sulphadiazine lipid emulsion for this study.

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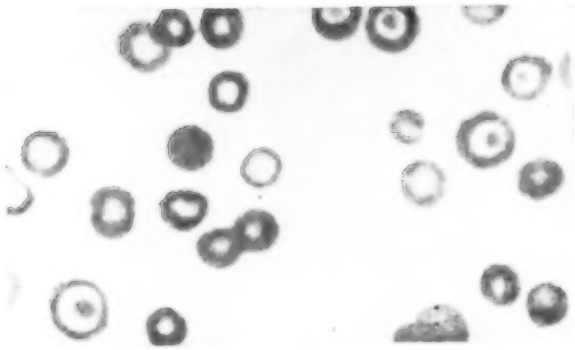

Fig. 2—Peripheral blood smear showing numerous target cells (Leishman's stain × 900)

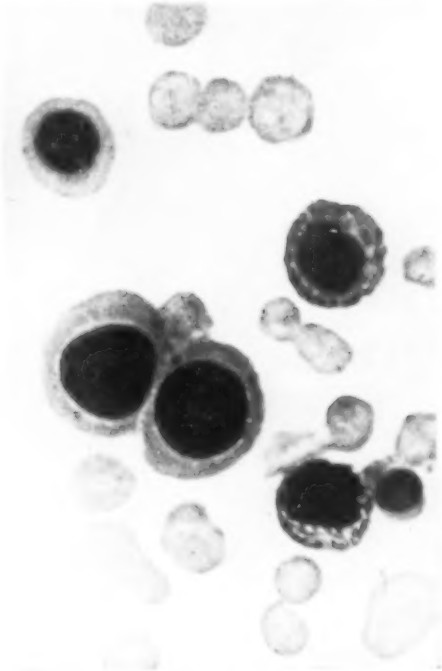

Fig. 4—Bone marrow smear showing two macro-normoblasts in the central part. The peripheral blood showed macrocytic hypochromic anaemia (Leishman's stain × 900)

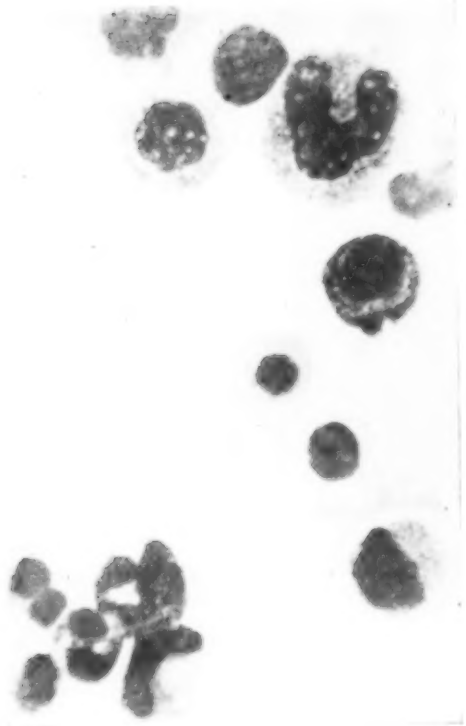

Fig. 5—Bone marrow smear showing one giant metamyelocyte (Leishman's stain × 900)

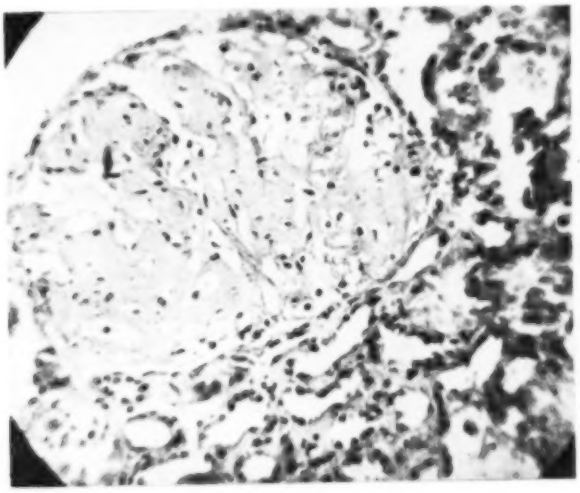

Fig. 1—Microphotograph showing masses of amyloid material in the glomerulus (H & E × 240)

SENGUPTA AND AIKAT—*Anaemia in Cirrhosis of the Liver (p. 407).*

MATHUR AND SRIVASTAVA—*Fatal Complication with Prednisolone Therapy in Nephrotic Syndrome (p. 414).*

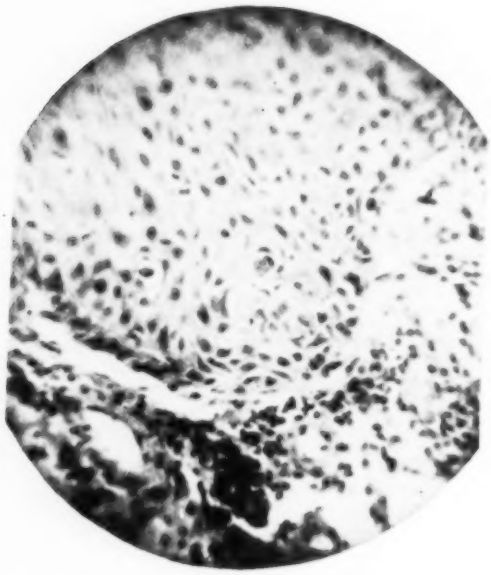

Fig. 1—Showing hyperplasia of the epithelium, the intact basal layer and premature keratinisation in the prickle cell layer. ×450

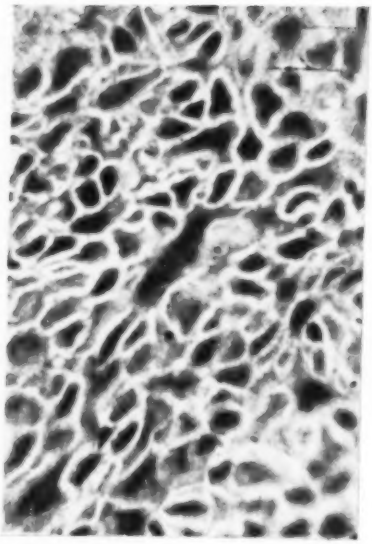

Fig. 2—Showing cellular pleomorphism, hyperchromatism of the nuclei and a 'monster' cell in the prickle cell layer. ×1000

SATYENDRAN AND REDDI—*Intra-Epithelial Epithelioma (Bowen's Disease)* (*p.* 424).

Fig. 1—Skiagram taken 20 minutes after administration of the dye showing the kidney and ureter on the right side

Fig. 2—Skiagram taken after barium enema showing the tumour lying lateral to the descending colon which is pushed to the right beyond the middle line.

JOHN AND RAZAK—*Retroperitoneal Mesonephric Cyst* (*p.* 425).

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LAURENCE-MOON-BIEDL SYNDROME

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In the year 1866, Laurence and Moon reported four cases with hypogenitalism, obesity, mental deficiency and dwarfism associated with retinitis pigmentosa, occurring in members of the same family. This bizarre combination did not attract further attention till the beginning of the present century. Bardet (1920) suggested that the association of these various defects was an entity by itself. Biedl and Raab (1922), quoted by Soffer (1956), described two cases in one family and stressed that congenital defects, like deformities of the skull and atresia ani, could also occur.

Ever since the above descriptions, other cases have been reported and so far about 150 examples have been described in the world literature (Soffer, 1956). In India the condition has been sporadically reported by Kutumbiah and Abbu (1942), Nandi (1949) and Laha (1958).

The following case exhibited the classical features and therefore merits description :

Case Report

Roshan Lal, 20, male, from Armitsar (Punjab), was admitted in the Rajendra Hospital, Patiala in November, 1958. He was considered to be normal at birth but his milestones were retarded ; he started speaking at the age of five. He was sent to school at nine but was too backward to carry on further study. He complained of nightblindness at the age of seven. About the same time he was noticed to be getting obese. No family history in other sibship was available.

Physical examination revealed an obese individual 172 pounds in weight and 5 feet and 1½ inches in height (Fig. 1). The fat distribution was more prominent about the shoulders, breasts and hips. Hair was sparse in the axilla and pubes and the penis was of small size. He had seven fingers and six toes (polydactyly) with syndactylism (Fig. 2). The patient exhibited behaviour disturbances and the higher cerebral functions were affected.

Fundus examination showed a deposition of coarse brownish pigment diffusely scattered all over. His visual acuity was poor and he could barely see at night. No other neurological abnormality was detected. The cardiovascular system revealed nothing abnormal. Blood pressure was 120/86 mm. Hg. The examination of the lungs and abdomen showed no abnormality.

FIG. 1

FIG. 2

The routine laboratory investigations were within normal limits. The basal metabolic rate was plus 9 per cent. The urinary 17-keto-steroid excretion in 24 hours was 5·1 mg. The skiagram of the skull did not show any evidence of increased intracranial tension and sella turcica was normal. Fasting blood sugar was 76 mg. per cent.

Comments

The syndrome known after Laurence, Moon and Biedl is very rare. Cockayne *et al* (1935) stated that it had not been described in Negroes, Mongols or any of the races in India.

The complete syndrome consists of obesity, hypogenitalism, sparse growth of facial, axillary and pubic hair, dwarfism, soft-textured skin, retinitis pigmentosa, polydactylism, mental deficiency and a positive family history. However, the syndrome may occur in an abortive form and in only about 50 per cent of the patients are obesity and hpogenitalism present together. The case under report showed most of the above mentioned features except that a family history of similar illness was not present. The investigations in him were essentially normal. The urinary 17-ketosteroid excretion was low (5·1 mg. in 24 hours). Roth (1947) described 4 cases in whom urinary 17-ketosteroid excretion varied from 3·2 to 6·3 mg. in 24 hours.

The chief interest of this syndrome is its aetiology which is not precisely known. Because of the familial incidence, genetic inheritance is considered to be the most likely cause. Sorsby *et al* (1939) suggested that the syndrome was determined by two recessive genes in the same chromosome or that it was dependent on some chromosome error such as dislocation or translocation. The other view is that two separate genes determine the syndrome, one accounting for the ectodermal defects in the hypothalamus and retina and the other determining the various skeletal deformities.

Summary

A typical case of Laurence-Moon-Biedl syndrome from Patiala (Punjab) is described.

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INTRA-EPITHELIAL EPITHELIOMA (BOWEN'S DISEASE)

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It is not uncommon for Bowen's type of dyskeratosis to occur in the corneal, limbal or bulbar epithelium, the limbal region being the commonest site. The disease is usually unilateral and appears clinically as a reddish granulomatous or gelatinous mass. Long-standing inflammation is a predisposing factor in its causation. One case is reported to have begun in a trachomatous pannus and another in an injured eye after an interval of many years (McGavic, 1942). According to Reese (1953) it is a more common form of epithelioma of the conjunctiva and cornea than the basal and squamous cell types. It may progress to a frank squamous cell carcinoma. Complete surgical excision is the usual treatment and gives excellent results. X-radiation is not satisfactory.

Case Report

Mr. S. H., Male, aged 44 years, presented himself at the out-patient department on 16-2-1959 with the complaint of irritation and lacrimation in the right eye since 2 months.

The vision in both eyes was 6/6. On external examination, there was a smooth gelatinous flat mass 3 mm. × 3mm. × 1mm, not freely movable, present on the medial aspect of the right eye. There was also a pterygium present on the lateral aspect of the limbus in the same eye. The left eye was normal. The case was clinically diagnosed as papilloma at the limbus.

The growth was excised on 18-2-1959 and sent for histopathological examination.

Histopathological report—"The markedly hyperplastic epithelium has an intact basal cell layer. The hyperplasia is entirely intraepithelial. The basal cells have lost their morphological characteristics and their regular arrangement. In the prickle cell layer cellular variations in size and shape, loss of polarity, premature individual cell keratinisation, monster cells, and a few mitotic figures are evident. This is an intra-epithelial epithelioma of the limbal region". (Figs. 1 and 2 *vide* Plate).

The postoperative period was uneventful and the patient was asked to report if there was any recurrence of the condition or persistence of the symptoms for which he came to the hospital. He has not reported afterwards and is presumed to be relieved of his trouble.

In reporting this case of Bowen's disease the difficulties and importance of recognising this disease clinically and the need in every case for histological examination is stressed.

ACKNOWLEDGMENT

Our thanks are due to Dr. P. Ramchander, Professor of Ophthalmology and Superintendent, Sarojini Devi Hospital, and Dr. P. Siva Reddy, Additional Professor of Ophthalmology, for permission to report this case.

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RETROPERITONEAL MESONEPHRIC CYST

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Standard text-books do not deal with retroperitoneal mesonephric cyst in detail and on account of its rarity we report the following case.

CASE REPORT

A woman, a tea estate labourer, aged about 40 years, was admitted to the Estate Hospital on 27-9-57 with the complaint of a tense swelling on the left hypochondriac region. She was a multiparous woman with 6 children, all normal confinements. The last child 3½ years old was normal. Her history was that for 2½ years, she had been feeling a heaviness on the left side of the abdomen without any evidence of tumour. But for the past 6 months she had been noticing a swelling there. This was growing in size and made her come to the hospital. There was no other relevant history. Her periods had been very regular. There were no urinary complaints such as haematuria, painful micturition, etc., at any time.

On examination, she was a well-built and healthy woman. A rigid, tense, round, non-tender mass, of the size of a small football, slightly movable in the horizontal position but not vertically, was found in the left hypochondriac region extending from the costal margin to the umbilicus. A finger could be passed between the tumour and the costal margin. Abdomen was soft. No other masses were felt anywhere. No palpable glands were found in the body. P. V. examination showed the uterus and ovaries to be normal. There was no bulging of the fornices.

Investigations—Hb. 65 per cent, the blood picture was otherwise normal. E.S.R. 37·5 mm. per hour. Repeated examination of the urine revealed no abnormality. Stool examination did not show anything abnormal. A plain x-ray of the abdomen showed an opaque shadow well-demarcated in the left kidney and lumbar region.

I. V. pyelogram—After 10 minutes: The demarcation of the opaque shadow was not clear. The left kidney and ureter were not visualised. The right kidney and ureter were visualised.

After 20 minutes: the demarcation of the opaque shadow was not clear. The left kidney and ureter shadow could not be seen. The right kidney and ureter were visualised (Fig. 1, *vide* Plate).

Barium enema—The descending colon was pushed to the right beyond the middle line. The shadow of the tumour was clear and well demarcated lateral to the descending colon (Fig. 2, *vide* Plate).

Operation—On 25-10-57 under spinal anaesthesia she was operated by a left paramedian incision. The transabdominal route was selected because of the size of the tumour. On opening the abdomen, a tense retroperitoneal cystic swelling was found to which the descending colon was seen adherent on the medial side. It appeared highly vascular and reflection of the peritoneum was found difficult. A needle was put in to find out the nature of the fluid and some pale coloured clear fluid was drawn. The cyst was then emptied by suction, 5 pints of fluid having been removed. The peritoneum was then incised and the cystic wall could be easily removed. The left kidney was palpated and found to be normal. This tumour was anterior to the kidney in the retroperitoneal fat. The retroperitoneal space was drained, the peritoneum was closed. Before closure the abdominal viscera was examined and everything including the pelvic organs was found normal. The recovery of the patient was uneventful. She was discharged on the 10th day.

The fluid in the cyst when examined was found to be pale in colour, neutral in reaction with a specific gravity of 1010. It did not contain any albumin or sugar but abounded in pus cells.

The cyst was sent for section and the pathological report read as follows: "The cyst wall shows glandular elements lined by tall columnar epithelium. The cells contain vacuoles probably mucin and the nuclei are situated towards the basement membrane. The appearances are suggestive of (1) serous cystadenoma of the ovary and, (2) a tumour related to the manner of embryological formation of the kidney. If there is no primary tumour in the ovary, the second alternative has to be thought of."

JOURNAL OF THE
INDIAN MEDICAL ASSOCIATION

CALCUTTA, NOVEMBER 16, 1959

ANTI-SNAKE-VENOM SERUM

A recent world-wide survey conducted by the World Health Organisation has indicated "that the number of humans bitten annually by snakes (poisonous and non-poisonous) in India may be of the order of about 200,000"[1]. Varying quantities of anti-snake-venom serum would be required for treatment of all these cases.

No doubt, many of the victims are bitten by non-poisonous snakes. However, more frequently than not the culprit escapes, and absolute reliance on the fang marks or development of signs of venom action may be hazardous. Even if the snake was caught or killed, its identification needs availablity of an expert in the immediate vicinity. Were it identified as a nonpoisonous snake, the victim is not necessarily saved. Not infrequently a person bitten by a non-poisonous snake suffers extremely from fear which develops severe shock and may even culminate in death. A small quantity of antivenin, like the charlatan's charm, may be useful in such cases.

Thus, all cases of snake-bite should be considered as potential requirers of antivenin in different quantities. It is, therefore, imperative that provision should be made in this country for making available the required quantities of antivenins, and perhaps an excess too, to meet all demands.

However, mere availablity in large quantities of a product labelled anti-snake-venom serum is not enough. The two essential criteria of a useful antivenin are that (1) it must be highly potent, and (2) it must be able to neutralise the venoms of the poisonous snakes of the region. These two essential criteria must be satisfied by all antivenins used for combating snake-bites.

The first criterion does not seem to be satisfied by all antivenins used in India. A recent study[2] on the potency of certain batches of antivenins, issued for use in man, revealed that some of them could neutralise only insignificant quantities of cobra venom. Such sera of remarkably poor potency could by no means save a victim effectively bitten by a cobra. Nevertheless, in the prevailing chaos regarding the preparation and assay of anti-snake-venom sera, nothing could be done to stop issuance of such useless sera.

In the matter of preparation and issue of antivenins extreme divergence of practice is still met with. Haffkine Institute, Bombay, prepares a polyvalent antivenin neutralising the venom of cobra, Russell's viper, krait, and saw-scaled viper and issues it in the freeze-dried state, while the Central Research Institute, Kasauli, prepares a bivalent antivenin neutralising the venoms of

[1] SWAROOP, S. AND GRAB, B.—*Bull. Health Org.*, **10**: 35, 1954

[2] SARKAR, J. K. AND LAHIRI, D. C.—*J. Indian M. A.*, **33**: 355, 1959

cobra and Rusell's viper only, and issues it in the liquid state.

The levels of claimed potency also differ. The Haffkine Institute, for instance, considers the antivenin to be satisfactory if one ml. of the serum neutralises 0·6 mg. of cobra venom while the Central Research Institute, Kasauli, issues such antivenins as would neutralise 2·0 mg. of cobra venom. Commercial preparations are claimed to be able to neutralise much smaller quantities, while actually they neutralise insignificant amounts, if at all, as was revealed by the study referred to above[3]. In several other countries, where anti-snake-venom sera are prepared, the methods of assay and the limits of potency vary[3]. Considerable confusion thus prevails in the manner of dispensation of such a life-saving drug as the anti-snake-venom serum.

Efforts of bringing order amidst the prevailing confusion have been made from time to time by requesting the Expert Committee on Biological Standardisation, World Health Organisation, to explore the possibility of the establishment of standards for control of antivenins. A recent review[4] of the present knowledge on this subject has indicated its complicated nature. Nevertheless, it has been the general consensus of opinion that cobra venom is reasonably homogenous to permit establishment of an International Reference Preparation of Naja (cobra) antivenin[5].

It has been suggested by some experts[6, 7] that assaying an antivenin in terms of a reference preparation of the antivenin is a much more dependable method than expression of its potency in terms of quantities of the venom neutralised per ml. of the antivenin. The experiments conducted recently by Sarkar and Lahiri[3] also support this contention. Indeed, expression of the potency of an antivenin in terms of the quantities of venom neutralised per ml. of antivenin is so anomalous that it would be difficult to control an unscrupulous manufacturer of antivenin on the basis of such checks. Perhaps, the deviations from the claimed potency of the products tested in this study[3] may be partly explained by this anomaly. However, that would not be all the explanation. Unless, there is provision for rigid State control of the essential biological products, the issue of products of low potency would not be eliminated.

The health authorities of a State should assume the responsibility of ensuring the availability of adequate quantities of anti-snake-venom serum to meet all demands, as well as of enforcing rigid control over the preparation and potency of this product. Spurious antivenins deprive victims of snake-bite from the protection offered by potent preparations. Unrestrained issues of faked antivenins not only endanger the safety of the afflicted but also ruin the reputation of the drug—a reputation which has been built up so very well through painstaking research.

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CURRENT MEDICAL LITERATURE

Apresoline and Serpasil in Pre-Eclamptic Toxaemia

MENON, M. K. K., Vohra, S. and Karamjit Singh (*J. Obst. & Gynaec. of India*, **9** : 285, 1959) from the Institute of Obstetrics and Gynaecology, Government Hospital for Women and Children, Madras, from an analysis of the results of treatment of 100 cases of pre-eclamptic toxaemia with apresoline and 183 cases with serpasil observe :

These cases are divided into grade A (mild) and grade B (severe). 50 cases of grade A were treated with apresoline tablets orally 50 mg. as initial dose increasing by 50 mg. daily according to response and not more than 400 mg. being given in 24 hours. 60 cases of grade B were treated with 20 mg. apresoline intravenously repeated if necessary twice daily and after stabilisation continued with oral apresoline. 58 cases of grade A were treated with 1 mg. of serpasil 3-6 times a day orally and 75 cases of grade B were treated with injections 2·5-5 mg. initially repeated every 4-6 hours.

The results were assessed as "good" response "fair" and "poor" response.

In grade A cases with oral apresoline "good" response was obtained only in 30 per cent of cases and with oral serpasil in 60·4 per cent. The control of blood pressure and foetal salvage rate was in no way superior to that obtained without any hypotensivrs.

60 cases of severe pre-eclamptic toxaemia were treated by apresoline injections. The blood pressure dropped to almost normotensive level in 75 per cent of cases within 3 hours of injections and was maintained at this level or appreciably hypotensive levels on an average for 14·6 hours. The drop in blood pressure begins within a few minutes of the injection. Good response was obtained in 60 per cent of cases, 30 per cent showed fair response and it was poor in 10 per cent. Using serpasil injections in grade B cases good response was obtained only in 21·3 per cent, fair in 36 per cent and poor response in 42·7 per cent.

Tachycardia was observed in all cases treated with apresoline. Side-reactions giving rise to any concern were not met with in either series.

There seems to be some improvement in prematurity rate and perinatal mortality rate when apresoline is used intravenously in toxaemia. But more cases have to be done to confirm this finding. For immediate significant reduction of blood pressure intravenous apresoline is useful, especially in labour and in imminent eclampsia. We are not impressed with the value of serpasil in the management of pre-eclamptic toxaemia.

Intravenous Oxytocin Drip in Treatment of Inefficient Uterine Action

GOLDMAN, L. (*J. Obst. & Gynaec. Brit. Emp.*, **66** : 382, 1959) from the Department of Obstetrics & Gynaecology, University of Natal and King Edward VII Hospital, Durban in giving the summary of a review of 300 cases observes :

Three hundred consecutive cases of inefficient uterine action, representing less than 2 per cent of the total number of deliveries over the same period, were treated by the intravenous oxytocin drip. Full dilatation within 12 hours was reached in 85·7 per cent of the cases. The perinatal loss, excluding those cases in which intrauterine death of the foetus had taken place before starting the drip, was 31 per thousand. Caesarean sections were performed in only 3 per cent of the cases.

Analysis of the failures indicates that the duration of cervical stasis is the most important single feature determining a good response to the treatment. High heads, posterior positions, malpresentations and incoordinate uterine action offer no contraindication to the successful and safe use of the drip.

The literature on rupture of the uterus associated with intravenous oxytocin drip therapy has been reviewed, and no case of rupture of the uterus occurring in a primiparous patient has been discovered. Three cases of rupture of the uterus associated with the use of pitocin for the induction of labour are reported. All of these were multiparous.

The only valid contra-indication to oxytocin drip therapy is the danger of rupture of the uterus in the multiparous patient. It is suggested that a more dilute solution should be employed in these cases and administration should be extremely cautious. Oxytocin should not be used in a suspect uterus.

Inefficient uterine action has been defined in terms of cervical stasis, and a plea is made for a therapeutic trial of oxytocin in all primiparous patients with cervical arrest at 2 or more fingers dilatation for longer than 6 hours.

Oxytocin Intravenous Drips in Missed Abortions

LONDON, J. D. O. (*J. Obst. & Gynaec. Brit. Emp.*, **66** : 277, 1959) of Simpson Memorial Maternity Pavilion and Royal Infirmary, Edinburgh, in reporting on the results of treatment of 18 cases of missed abortion with high concentration pitocin intravenous drip observes that this technique offers a reliable, rapid and soft method of management of missed abortion.

Recurrent Jaundice of Pregnancy

SVANBORG, A. AND OHISSON, S. (*Am. J. Med.*, **27** : 40, 1959) from the Medical Clinic II and the Department of Pathology, University of Gothenburg, Gothenburg, Sweeden, give in the following lines the summary of their observations on the clinical study of 22 cases of recurrent jaundice :

The disorder differs clinically and in other manifestations from other types of jaundice occurring during pregnancy.

Seven patients showed a drop in the proconvertin-prothrombin levels of the serum and a potential general tendency to bleeding. If this complication can be diagnosed in time, haemorrhage can be prevented by the parenteral administration of vitamin K, which results in a rapid return to normal of the proconvertin-prothrombin level.

All patients who had had jaundice during a prior pregnancy had a recurrence of the disease in a subsequent pregnancy. The clinical picture during the re-

currence was strikingly similar to the previous attack.

Liver biopsy was performed during periods of jaundice in five patients. The histologic observations were dominated by signs of biliary stasis with dilatation of the bile ducts and accumulation of masses of bile pigment. No residual liver changes were observed in two patients investigated again after delivery and after the clinical picture had become normal.

The prognosis appears to be good; residual symptoms have not been demonstrated even after three recurrences.

The cause of the disease seems to be impairment of bile flow. The pathogenesis of this biliary stasis is not known.

Evaluation of Low Rupture of Membranes As Method of Induction of Labour

Eton, B. (*J. Obst. & Gynaec. Brit. Emp.*, **66**: 462, 1959) from the Department of Obstetrics & Gynaecology, United Cambridge Hospitals in giving a review of a continuous series of 500 inductions by low rupture of the membranes observes:

In cases of toxaemia the shorter induction-delivery interval renders rupture of the forewaters preferable to high rupture. The foetal mortality increased with the induction-delivery interval, but in many cases there were other more definite hazards to the life of the child.

For the treatment of disproportion, induction seems to have a definite place in multigravidae.

In cases of prolonged pregnancy the diagnostic criteria of postmaturity may be controversial. Examination of the liquor amnii may indicate whether the foetus is in danger. In such patients no liquor may be obtained with the cannula method.

In patients with unstable presentation low rupture of the membranes is more likely to make the presenting part engage, but there may be a greater risk of cord prolapse than with high rupture of the membranes.

In cases of presentation of an extended breech low rupture of the membranes is satisfactory, but with a high flexed breech there may be a place for high rupture.

There is no maternal mortality directly attributable to the induction method of rupture of the forewaters. The same cannot be said in the case of high rupture of the membranes.

There was no evidence that rupture of the forewaters entails a higher risk of uterine infection or foetal mortality than rupture of the hindwaters.

Foetal Wastage in Twin Pregnancy

Tow, S. H. (*J. Obst. & Gynaec. Brit. Emp.*, **66**: 445, 1959) from the Royal Maternity Hospital, Belfast, gives in the following lines the summary of his observations:

A foetal mortality of 12·3 per cent is demonstrated among a series of 408 consecutive twin pregnancies.

Antenatal, intranatal and postnatal risks of twin pregnancy, in large measure associated with gross prematurity, contribute to this high mortality.

Antenatal care and bed rest in hospital are shown to confer striking benefits in reducing maternal complications and prolonging gestation, thus leading to a reduction in prematurity and foetal wastage.

Early attendance by pregnant women, accurate diagnosis and careful antenatal supervision are essential. In order to prevent onset of very premature labour, bed rest in hospital from the 30th week is suggested.

Only one-third of the patients delivered both babies spontaneously. The need for operative assistance in the remaining two-thirds makes it imperative for all twin pregnancies to be confined in hospital.

The second twin is specially susceptible to perinatal death on account of labour hazards arising from malpresentation and disturbed uteroplacental circulation following the birth of the first twin.

In order to reduce the risk of such death, rupture of the membranes within 10 minutes followed by prompt and skilled delivery is advocated.

Staphylococcic Pneumonia

Ede, S., Davis, G. M. and Holmes, F. H. (*J. A. M. A.*, **170**: 638, 1959) from the U. S. Naval Hospital, Great Lakes, Ill. write that an attempt to correlate the incidence of primary staphylococcic pneumonia with the use of antibiotics revealed that the incidence is apparently no greater now than prior to the development of antibiotics. Thirty-four cases of primary staphylococcic pneumonia and two cases of secondary staphylococcic pneumonia observed at the U. S. Naval Hospital, Great Lakes, Ill., since 1955 were included in the study. Chloramphenicol (chloromycetin) plus erythromycin, novobiocin plus erythromycin, or a combination of all three seemed to give the most favourable results in the medical management of the disease. A high incidence of complication was seen with this form of pneumonia.

Close cooperation between the surgeon and internist should be maintained, and early surgical intervention is advisable when complications arise. The prompt institution of closed tube drainage with use of a large catheter (30 to 32 F.) for pyopneumothorax, empyema, and progressive pleural effusions has been instrumental in saving lives and reducing morbidity.

Secretion of Adrenaline by Adrenals in Reserpine-treated Rabbits

Kroneberg, G. and Schumann, H. J. (*Arch. exper. Path. Pharmak.*, **234**: 133, 1958, Ref. *German M. Monthly*, **4**: 104, 1959) from Pharmakol. Institute, Univ., Frankfurt, write:

Plasma was collected from the left adrenal vein of rabbits anaesthetised with urethane. When no reserpine was given adrenaline was secreted at a rate of between 0·065 and 0·13 μg/Kg/min.; noradrenaline was not detected. One half to 4 hours after giving 4 mg./Kg. reserpine i.v. the adrenaline content of the plasma was usually 2 to 3 times higher than previously. After splanchnicotomy reserpine did not increase adrenaline secretion. 20 hours after the same dose of reserpine the adrenal gland was almost completely depleted of adrenaline, but the secretion of adrenaline was only reduced to about 50 per cent of the control value. There was, however, no relation between the degree of depletion of the gland and the rate of secretion of adrenaline. In some experiments the adrenaline stores were

depleted although the increase in adrenaline secretion was only slight and transitory. The increased secretion does not, therefore, suffice to explain the depletion of the gland. Pretreatment with iproniazid, an inhibitor of monoamine oxidase, somewhat reduced the depletion caused by reserpine. The authors suggest that after large doses of reserpine some of the adrenaline liberated in the adrenal gland may be destroyed there by monoamine oxidase, and thus fails to reach the adrenal vein blood. The disparity between adrenaline secretion and depletion of the gland may also be due to an inhibitory effect of reserpine on adrenaline synthesis, since electrical stimulation of the splanchnic nerve increases the secretion without depleting the gland.

Heart in Friedreich's Ataxia

WAHI, P. L., SINGH, SURRINDER AND CHHUTTANI, P. N., (*J. A. Physicians of India*, **7**: 289, 1959) from the Department of Clinical Medicine, Medical College, Patiala in reporting on thirteen cases of cerebellar ataxia along with 19 uninvolved siblings who were studied for cardiovascular lesions clinically, radiologically and electrocardiographically observe:

Two patients showed abnormal findings: one had extrasystoles and coronary type RST-T changes in most of the precordial leads while the second had pulmonary stenosis.

Valsalva Manoeuvre As a Diagnostic Aid

IRVIN, C. W. (*J. A. M. A.*, **170**: 787, 1959) of Columbia, S.C. writes:

The test for cardiac function consists of having the patient blow into a manometric system in such a way as to maintain an intrapulmonic pressure of 40 mm. Hg. for 10 seconds. During and immediately after this straining period the patient's blood pressure is noted. A four-phase response, consisting of a brief rise of 30 to 50 mm. Hg in systolic blood pressure, a gradual fall to resting level, a brief fall below resting level when the strain is released, and a secondary rise of 15 to 40 mm., is considered a normal response. The test was applied to 205 selected office patients. Positive results (abnormal responses) were obtained in 35. Of these, 14 showed clinical signs of congestive heart failure and 11 had significant heart disease.

Bentonite Flocculation Test In Rheumatoid Arthritis

DELTOMO, R. A., SMITH, R. T., KRON, K. M., HERMANN, I. F. AND CLAPPIER, M. H. (*J. A. M. A.*, **169**: 315, 1959) from the Department of Medicine, Section of Rheumatology, Benjamin Franklin Clinic and Pennsylvania Hospital, Philadelphia give in following lines the summary of their observations on bentonite flocculation test in 160 cases of rheumatoid arthritis.

A total of 160 patients' serums have been subjected to the bentonite flocculation test as devised by Bozicevich *et al* (*Proc. Soc. Exper. Med. & Biol.*, **97**: 180, 1958). The test is easier and less expensive to perform than any of the heretofore described serologic tests for rheumatoid arthritis.

Eighty-three per cent of 48 patients with definite rheumatoid arthritis and 33 per cent of 12 with probable rheumatoid arthritis had positive findings in the flocculation test. These results are comparable to those in previously reported serologic tests with more difficult and expensive procedures.

Only 2 of 19 patients with rheumatoid spondylitis had positive results, and only in the lowest positive titre, although the majority of these patients had subsequently developed peripheral joint involvements. This, again, stresses that these two conditions probably have different aetiologies.

Only 3 of 35 patients with other rheumatic conditions exhibited positive results, and 2 of these had had previous diagnosis of combined gouty and rheumatoid disease. Only one, then, a patient with systemic lupus erythematosus, had a true false positive reaction. Previous reports stated that 20 to 30 per cent of patients with this condition will give false positive reactions to serologic testing for rheumatoid arthritis, suggesting a possible relationship between the two conditions.

Of 48 hospital and clinic patients comprising a heterogenous group of conditions characterised by altered protein metabolism, only one gave a positive result. This patient had a mild type of hypertension with no evidence of a rheumatoid state, but further studies and observations are indicated.

This report substantiates the conclusion that the bentonite flocculation test is the most recent, useful advance in rheumatoid serology and is an additional step toward the development of an accurate, simple, inexpensive, and dependable laboratory aid for the diagnosis of rheumatoid arthritis.

Clinical Evaluation of Paper Electrophoresis

GOLDBLOOM, A. A. AND BOYD, L. J. (*Am. J. Gastroenterol*, **30**: 581, 1958) from the New York Medical College—Metropolitan Medical Centre (Metropolitan Hospital Division), New York, in finding out a correlation between the change of the electrophoretic serum lipoprotein patterns and the chemical serum lipid determinations in patients with liver disease observe:

The electrophoresis showed an increased gamma, decreased alpha-1 and beta components. The alpha-2 component was of no clinical aid or significance.

The above lipidogram changes were similar to previously published findings of the protein paper electrophoresis in liver disease.

Increased, decreased, or normal serum lipid partitions were accompanied by the same lipoprotein electrophoretic changes.

In liver disease, the processes go from carbohydrate to fat metabolism because of an inhibitor factor. The inhibitor factor makes the cholesterol nonmobile or demobilised, combined with neutral fats, resulting in an increased gamma lipoprotein component, with a normal cholesterol (chemical).

The lipidogram will indicate the type, mobility, or usefulness of total cholesterol if decreased.

Cholesterol metabolic disturbance should be checked by other methods when there is evidence of the inhibitor factor.

In liver diseases, phospholipids are utilised, since the metabolism of fats and not carbohydrates takes place.

Phosphorus or phospholipids may be normal by chemical procedures—yet, the lipidograms will reveal that the phospholipids are not the proper nor useful type for utilisation.

Chemically, normal or high cholesterol may be shown, while paper lipoprotein electrophoresis (lipidogram) will reveal a deficiency in the type of cholesterol.

The filter paper lipoprotein electrophoresis (lipidogram) as with the protein fractionation produced a better understanding of the disturbed physiology and underlying pathology in the patients with liver disease.

Since filter paper electrophoresis is accurate, rapid, inexpensive, and simple, it should be a procedure in the clinical laboratory because of its aid to the clinician. (*Authors' summary*).

Haemolytic Reactions to Drugs

Haemolysis is a well-recognised complication of the administration of some drugs, and phenylhydrazine was for long used in treating polycythaemia on account of this action. If haemolysis is severe haemoglobinuria may occur and anaemia develop, but if it is mild the process may not be noticed unless sensitive methods of detection are used. Phenacetin has not been incriminated as a cause of haemolysis until recently, but three reports from Switzerland strongly suggest that such a complication may sometimes be overlooked.

In the first of these papers Marti (*Schweiz. med. Wschr.*, **88** : 1054, 1958) describes 70 cases of chronic phenacetin overdosage in patients who were taking between 1 and 10 g. of the drug daily. Half of these had haemoglobin concentrations of less than 10 g. per cent, and of these only one-fifth were uraemic, so that renal failure could not have been the cause of the anaemia in all cases. The fact that significant reticulocytosis was present in 15 of these patients suggested that the anaemia was a result of either chronic blood loss or haemolysis. Unfortunately blood loss was not excluded in all the patients, but it is probably fair to conclude that haemolysis was the essential cause of the anaemia if evidence from the other papers is taken into consideration. In view of the variability in the occurrence of anaemia the author concluded that there was an individual factor controlling its occurrence and that its degree of severity was not related to the dose of drug. In a further paper Miescher and Pletscher (*Ibid*, **88** : 1056, 1958) determined the survival rate of red cells by the radiochromate method in patients taking 1-2 g. of phenacetin daily. Red-cell survival was reduced in these patients, but not to the extent that would ordinarily have caused anaemia in a patient with a marrow capable of normal compensation. That the reduction in the life span of red cells was due to irreversible cell damage was shown by the shortened life span of the same cells when transfused into healthy drug-free recipients. This fact and an increased urinary excretion or radioactive chromium led to the conclusion that intravascular haemolysis was occurring. The authors point out that, although the rate of haemolysis would not normally cause anaemia, it might well do so in patients with chronic diseases in whom marrow activity was depressed, these being patients most likely to be taking phenacetin continuously. The same workers (*Ibid*, **88** : 1214, 1958) have studied the effects of phenacetin and its derivatives on the life span of red cells in rabbits and dogs The doses of drugs used were much greater than the therapeutic doses, but they found that phenacetin, acetamidophenol ("panadol"), and *p*-phenetidine all depressed red-cell survival to an equal extent, suggesting that acetamidophenol, the *in vivo* metabolite of phenacetin which is usually claimed to be less toxic, was not devoid of this damaging effect on red cells.

The evidence from these papers does suggest that haemolysis may occur as a toxic effect of phenacetin, but more work is required to find out if this is a frequent complication of long-continued phenacetin administration in ordinary therapeutic doses. There is no evidence of how the haemolysis occurs. Two processes are known to be involved in haemolytic reactions to drugs. In the first the drug appears to act as a hapten and combines with red cells to render them antigenic. This is probably the mechanism in haemolysis complicating the administration of quinidine (*Freedman et al, Am. J. Med.*, **20** : 806, 1956) and perhaps quinine, methoin (*Snapper et al, Ann. Int. Med.*, **39** : 619, 1953), and stibophen (Harris, *J. Lab & Clin. Med.*, **47** : 760, 1956). More often thrombocytopenia occurs, but the mechanism causing this appears to be the same. The second process has received much attention since Dern and his co-workers (*J. Lab. & Clin. Med.*, **45** : 30, 1955) found that negroes who developed haemolytic anaemia when given primaquine showed an enzyme defect in their red cells which interfered with the normal mechanisms maintaining glutathione in its reduced form. The enzyme was later found to be glucose 6-phosphate dehydrogenase. This defect has now been found in red cells of patients developing haemolysis after taking a number of different drugs; these include sulphonamides and para-aminosalicylic acid, nitrofurantoin, and vitamin K analogues. The enzyme defect appears to be transmitted as a sex-linked character and can easily be detected by the glutathione stability test. The way in which glutathione oxidation interferes with red-cell survival remains to be determined. Haemolysis occurring after the administration of other drugs such as acetanilide, glucosulphone, neoarsphenamine, and phenylsemicarbazide ("cryogenine") has also been reported, but the mechanisms leading to this complication have not yet been defined. (*Annotation, Brit. M. J.*, **1** : 841, 1959).

Prednisone in Mumps Orchitis

MONGAN, E. S. (*Am. J. M. Sc.*, **237** : 749, 1959) from the Communicable Disease Service, William Beaumont Army Hospital, El Paso, Texas, gives in the following lines the summary of his observations on the results of treatment of mumps orchitis with prednisone :

A double-blind, forward-looking study comparing the results of therapy in patients with mumps orchitis treated either symptomatically, or with placebo, or with prednisone revealed no differences in the course of the disease in the three groups.

CURRENT TOPICS

FAMILY PLANNING

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President, Indian Medical Association

The Medical Council of India has, in a recent circular, recommended that instructions in family planning should be an integral part of undergraduate medical education. The superficial observer might perhaps interpret this, as misguided enthusiasm of the Council to fall in line with the Government which has placed family planning in the forefront of its programmes. The promotion of the health of the nation is among the fundamental objectives of all Governments. For that a healthy balance between national resources, immediate as well as potential, of the State and its population is necessary. And when the State comes to the final conclusion, on expert advice, that it would be impossible to improve living standards, especially the state of nutrition, without limitation of population, medical educationists cannot ignore it, because promotion of national health, is the foremost concern of the medical profession. Therefore the inclusion of Family Planning in Social Medicine is appropriate.

Growth of Population

Prof. Julian Huxley, the well-known scientist, said recently that the present age is not so much the age of sputniks or rockets as of mounting population. He also stated that we have not the resources to feed half the present population and educate a third. The significance of these warnings will become clear if we look at the changes in the pattern of population growth, through centuries, as may be seen from the following table.

Year	World population
A.D. 30	250 million
1650	545 million
1750	728 million
1850	1171 million
1950	2400 million
1954	2642 million

It may be seen from the above that till 1650 the annual rate of increase was only 0·06 per cent.

From 1650-1850 the rate was 0·5 per cent. From 1850-1950 the rate was about 1 per cent. From 1950 the rate has been 2 per cent.

Now the world population is estimated to be 3000 million and the annual addition is 60 million. At the present rate of growth, the population by the end of the present century will rise to 6000 million and in the next 30 years it will exceed 10,000 million. It is evident that in about 60 years there will be a three-fold increase in world population, if the present trends continue.

Checks on Growth

The causes responsible for the almost negligible increase till the 18th century of the Christian Era can be divided into two main heads: (i) Natural, which man could not control or influence and (ii) Artificial or man-made. The most potent natural causes which checked the growth of population were famines and diseases.

Famines

These occurred not only in isolated parts of countries, but frequently entire countries were affected and occasionally they were of global character. To illustrate the devastation caused by famines a few instances may be cited. In B.C. 436 famine was so severe in Rome that the starving people in large numbers found relief in committing suicide, by throwing themselves into the rivers. The Potato Famine of Ireland (1846-47) killed hundreds of thousands and led to the mass emigration of the people to the United States. In 941, 1022 and 1033 entire provinces in India were depopulated and cannibalism is reported to have been widely prevalent. Ten million people died in Bengal in 1769-70, which was a third of the total population of the province. Even as late as 1901, 10 lakhs of people died of famine in India. Widespread famine of this magnitude used to occur in other countries also, especially in China and Russia. During the last world war according to official estimates 7 lakhs died of famine in Bengal (the unofficial estimate is well over 1 million).

Diseases

Visitations of epidemics of smallpox, cholera, plague, etc., used to make a clean sweep of large masses of people.

"Before the days of vaccination scarcely 5 persons out of a hundred escaped it (smallpox) and about a quarter of those who took it died", says Rosenau.

That means that prior to 1800 (Jenner's experiments in vaccination were in 1796) 24 per cent of the world population used to be killed by smallpox. In a single year, 1919-20, influenza killed 20 lakhs.

In India from 1904 to 1923 about 4 lakhs died annually from cholera and about 5 lakhs from plague. Other diseases like malaria and tuberculosis, although they seldom assumed the terrifying explosiveness of plague and cholera, were equally large-scale killers of man. For instance till recently malaria accounted for 15 lakhs of deaths in India every year and tuberculosis killed one every minute i.e. 5 lakhs annually. These four diseases (plague, cholera, malaria and tuberculosis) used to account for nearly 30 lakhs which was more than 50 per cent of the total births. This was true of several other densely populated and backward countries of the world. Plague, cholera and smallpox have been totally wiped out in many countries and in others they are well under check. Malaria and tuberculosis have been controlled and the campaign for their total eradication is in progress. That is how Indian death rate fell from 44 (1900) to 20 per 1000 in 1955.

Heavy maternal and infant mortality was also a significant contributory check on the growth of population.

Man-made Causes

Wars stand in the forefront of the causes for which man's wickedness and cupidity are responsible. The youth of nations from the beginning of history, was periodically sacrificed at the altar of perverted patriotism, religious fanaticism, or the right of survival. World War I is perhaps the most classical example of the colossal loss to life caused by war. In addition to 66 lakhs of deaths among military personnel 32 lakhs of civilians died from war—a total of 98 lakhs! Also the number of births in the belligerent countries were less by 126 lakhs in comparison with births during the prewar period! Wars, although fought on a global front only twice, have been among the most notable recurring historical events of countries. Each war ended with a reduction of population of the belligerent countries. Often it was the pressure of population that forced political gamblers to seek an escape in war.

Mass Migration

This is resorted to either to escape political or religious persecution or in search of a new land for better living or aggrandisement. In the migration of the Aryans from Central Asia to India, in the then conditions of travel a larger number of the very young and old as well as the weak and infirm would have fallen on the way. Till the beginning of the 19th century when more settled conditions were established large, and small scale exoduses were not infrequent occurrences. But the loss to human lives from such voluntary or enforced exodus, pale into insignificance, before the total decimation of entire races by colonial adventures as in South American countries which Froude the historian rightly condemned "as the enormous wickedness with which humanity has been disgraced."

Religious, Social and other Injunctions and Customs and Practices

The nomadic people found it impossible to carry more than one child and so abortion and infanticide were widely practised by them. "The pagan tribes of North when converted tried to bargain that they should retain the right to kill their children and infanticide continued to be widely practised in Japan till the beginning of the 19th century."

The Hindus extolled Sati as a virtuous act and banned remarriage of widows. Infanticide, particularly of female child, was widely prevalent, probably till the beginning of the last century. The *Mahabharata* illustrates in the most striking manner the evils of numbers. Hundred sons born to a mother (Kauravas) were so devoid of basic human qualities that there was divine intervention for their destruction by their cousins, who were only five in number!

References to contraceptive preparations and abortifacients are found in the ancient Egyptian, Islamic, Hindu and Chinese writings. There can be little doubt that crude methods to limit families to resources were practised all the world over, from very early days.

Celibacy was extolled by religions. The Catholic and Buddhist parents considered it their sacred duty to permit or persuade a son and a daughter to join the order of monks and nuns. The Nambudiris of Kerala, till recently, allowed only the eldest son of the family to marry from their own caste. Since Nambudiri women could not be married to non-nambudiris how this custom restricted the growth of the community is evident.

Renaissance and After

The change in population pattern commenced with the Renaissance in the 16th century. The increase since the 17th century is an index of the progressive scientific and cultural advancement of man. Famines ceased to kill in large numbers. Food production increased due to improvements in agriculture. There is generally enough for sustaining a substandard level of nutrition, even in densely populated backward countries. The development of communications, especially of aerial transport, has made it possible to rush food to areas threatened by famine even in the most isolated parts of the world. The growing faith in the brotherhood of man among civilised nations inspires them to mobilise resources and rush supplies to regions faced with famine. Scientific medicine has eliminated or reduced to manageable dimensions the former captains of death—smallpox, cholera, plague, malaria, etc. Modern maternity and child welfare schemes save millions of lives.

The world has become so closely knit by communications and cultural contacts that no megalomaniac in control of the destiny of a people, can unleash the dogs of war on his neighbours. There is a general conviction that war has ceased to be a solution to any problem, and with the armaments now available to the protagonists of conflicting ideologies, might lead to the extinction of the human race.

The natural and man made causes which checked the growth of population through the ages have been either totally eliminated or rendered more or less impotent and the population is growing at an accelerated pace.

The Evils of Overpopulation

Although the world knew, from ancient times, that it was dangerous if population outgrew resources, the most rational and convincing elucidation of that concept came from Rev. Thomas R. Malthus, in 1798. Malthus stated that while the population increased in geometrical ratio, resources increased in arithmetical proportion. This widening gulf between population and resources is the cause of most of the problems which overpopulated countries are facing today.

Undernourishment

The most important of these is the progressive fall in the level of nutrition. Diet surveys carried out in this country between 1935 and 1940 showed that the diets of the vast majority were qualitatively and/or quantitatively insufficient. Recent official reports show

that there has been no improvement since then. Malnutrition undermines physical and mental efficiency and a vicious circle is produced.

Deterioration of Educational Standards

It is admitted by everyone that educational standards have fallen and are falling. Twenty thousand children are born in Bharat daily, annually about 75 lakhs! At a conservative estimate 60 lakhs survive up to the school age. That means, the nation should have 20,000 primary schools annually (at 300 pupils per school) and about 4,000 secondary schools with a corresponding number of colleges. This is a physical impossibility. In a desperate attempt to educate children, schools are being established without adequate accommodation, staff, equipment and playgrounds. And another vicious circle sets in. Successive generations are taught in more ill-equipped schools by more incompetent teachers!

Mounting Unemployment

The millions who emerge from schools and colleges annually have no employment. Even graduates become attenders or bus conductors. Under-employment and unemployment are not new phenomena. Once there was a partial escape by supplying cheap labour to British colonies, to Ceylon, Malaya, etc. While the European nations sought relief to overpopulation by large-scale migration to conquered colonies as masters, China, India and Indo-China and other backward countries supplied slave labour for the exploitation of colonial resources.

It may be recalled in this context that the danger of overpopulation was dramatically brought to my attention by the shipment of thousands of half-starved miserable looking labourers to Malaya. That was in 1922. I was travelling to Malaya, where I was to work as Chief Medical Officer to the plantations of a District in the State of Johore. (I was there for seven years). The steamer in which I travelled was packed with underfed South Indian labourers, in rags. They were transported almost as cattle. That was happening every week. For the first time I realised the enormity of our population problem and how it had forced an ancient nation to seek some relief in this manner. What I saw in Malaya was even more humiliating. An Indian labourer got only 40 per cent of the wages which a Chinese labourer got—not out of racial discrimination, but because of lower output of work. The human organism adjusts itself, in a large measure, to sub-standard levels of nutrition, but as stated already it affects production capacity, intellectual as well as physical. If the cost of production is much lower in Japan and China than in India it is not due to sweated labour or poorer working conditions. It is because man for man the Japanese and the Chinese labourer have a far better economic value. If the protective tariff walls are demolished, our flourishing cotton mills will be strangled to death by the cheaper cloth with which Japan and China will be able to flood our markets. The progressive deterioration of physical and intellectual output is as dangerous to the future of the nation as growing unemployment. And let it also be remembered, that indigenous population is meeting the labour requirements to such an extent that Indians are no longer required in the old role of cheap labour or subordinate clerical staff. Nay, those who had gone before and worked hard to develop these countries are being slowly pushed out!

Undermining of Women's Health

Many births, at short intervals, is a recognised cause of deterioration of maternal health. That is why along with industrialisation and equal opportunities for work for women and men, family planning has been readily and voluntarily accepted in all advanced countries, including U.S.S.R., with a density of only 22 per square mile. Because women's health and efficiency are vital factors in national economy, promotion of their health by family planning is essential for national prosperity.

Threat to Peace and Progress

The expectation of life at birth, in different countries which may be seen from the following table, deserves serious attention.

	Expectation of life (males & females)	Birth rate per 100 1900-1950		Density per sq. mile
United States				
Whites	... 69·5	24	23	50
Coloured	... 66·0			
United Kingdom	... 69·5	27	16·7	53·2
Sweden	... 70	29	17·4	40
Norway	... 70·5	27	19·4	24
Australia	... 68·0	27	23	2·7
Newzealand	... 70·0	24	N	18·3
Japan	... 63	32	30	583
China	... 43	N	43	123
India	... 32	N	28	243

N—Not known.

	Comparative fall in birth-rate in 15 years	
	1940	1954
Japan	35	20
Rumania	37	20
India	30	35

(According to authoritative estimates Indian birth rate was 43 in 1955). Among 76 countries of the world for which the 1940—50 figures are available, India has the lowest expectation of life. Our birth rate is higher than most countries as may be seen from the same table. Low expectation of life with high birth rate means that younger age groups are more numerous than the more mature groups. The preponderance of the younger and more emotional and explosive groups, mostly frustrated by unemployment, is a grave threat to political security in countries having the right of adult franchise. Also the absence of an adequate num-

ber of experienced mature persons is a handicap to progress in all fields of national endeavour.

Solution by science—The tantalising prospect that scientific advance will soon find an answer to the growth of population is occasionally advanced by opponents of population control and scientific visionaries. They ignore the fact that notwithstanding the marvellous progress of science, a dangerously high proportion of the present world population is underfed, ill educated and unemployed. Therefore there is no justification whatever to expect a reversal of the present trends. When we are within sight of adequate nutrition and gainful employment for all a reorientation may be justified. At present we cannot be blind to what other countries, endowed with incomparably greater resources, are doing.

The density of population and the birth rate given in the preceding tables are revealing. In the United States of America, admittedly the richest country in the world with vast resources and with only 50 people per square mile, the birth rate is 23 per 1000. In Sweden with a density of only 24, birth rate fell from 29 in 1900 to 17 in 1950. Australia with a density of 27 has a decreasing birthrate, which is now 23. China has a larger birthrate than India, but has only 50 per cent of the Indian density. Great Britain has reduced her birthrate from 27 in 1900 to 16·7 in 1950. Japan reduced it from 35 to 20; and so did Rumania. Neither potential resources nor the possibility of their exploitation is an excuse to ignore immediate needs. No civilised nation has done that. They have been guided by certain clear considerations; i.e. standard of life and education and prospects of employment of the children. It is these considerations that have promoted millions of parents in all countries to adopt family planning without persuasion or coercion. And these considerations make the case for control irresistible.

Methods of Control—Natural and Artificial

While there is general agreement on the urgency of control, there are divergent views on methods. Some frown on artificial methods as being contrary to religion and culture. They advocate celibacy and abstinence and are inclined to tolerate 'safe period' union. Those who are keen on immediate effective check, doubt if tangible results will ever be obtained by so-called natural means. Also they argue that enforced celibacy, which is practised by certain religious sects is 'unnatural'. And from 35 years' experience, starting with appeals to religious and social leaders, to raise the age of marriage of women to 20 and interdict the marriage of the physically unfit, I doubt if the protagonists of natural methods are sincere in their protestations. Whatever that be, if in preventing and controlling diseases and raising expectation of life, artificial methods are justified, with equal validity artificial methods can be applied to limiting population to resources. The medical profession has special responsibility in this matter, because they are mainly responsible for eliminating or checking some of the powerful natural causes (diseases) which contributed to the natural balance between population and food.

Artificial methods—The adoption of artificial methods is therefore inescapable. They may be classified into two main heads depending upon whether or not fertility is permanently destroyed. Sterilisation operations belong to the first and contraception and abortion belong to the second group.

Oral contraception—A cheap and dependable oral contraceptive is the ideal to be aimed at. Although encouraging claims have been put forward by several investigators, it appears that this ideal is yet far off.

Mechanical and chemical contraceptives—These prevent the spermatozoa from contacting the ova, either by mechanical barrier or by chemical destruction. These methods have been widely practised, with good results, in the countries of Europe and America. Their advantage is spacing of pregnancies coupled with limitation. But under conditions of living obtaining in India, these methods are inapplicable to the lower income groups who constitute more than 75 per cent of the population.

Abortions—There is a growing volume of opinion, led by eminent doctors, that India should follow Japan and permit induction of abortion, on the written request of the husband and the wife. Medical ethics, forbid abortion except when the mother's life is in danger. But the Japanese people, famous for their practical wisdom, in their anxiety to limit the number of children to the income of the family, resorted to secret abortion to such an extent that it became a serious national problem. Eventually the Government was forced to pass the Eugenic Protection Law in 1948, by which induction of abortion was permitted on economic grounds also. And so the number of legalised abortions began to increase and by 1956 it was 1·2 million annually —nearly equal to the number of births! That was how Japan succeeded in reducing birth rate from 35 to 20 in about a decade. But this brief experience has shown that, although effective in checking the growth of population, abortion has certain disadvantages. In addition to the inherent threat to health, which might be serious in certain cases, abortion is quickly followed by pregnancy leading to two or even three abortions in one year. Repeated abortions of this kind undermine health and so the Government is now popularising other methods of control.

Sterilisation operations—Vasectomy and tubal ligation, if done properly, are fool proof methods of limitation. The objection to this is that procreative power is permanently lost and if a couple, for any reason, desires to have more children, there will be frustration. This defect weigh little against the enormous advantage that no other method is applicable to the lower income groups of this country. But a people steeped in apathy, with blind faith in the law of Karma, cannot be expected to come forward for operation out of sense of duty. These people are not however so devoid of basic intelligence, that they will refuse to respond, if facilities and encouragement be given for sterilisation. It is from this conviction that I have for some years advocated a graded tax on child birth, from the fourth birth. It is not intended that a new tax should be imposed on the poor people It will be the duty of the health

visitor, the midwife or the village officer in charge of birth registration, to notify every woman, as well as her husband, at the time of her third confinement (in case the two previous children are living and healthy) that a free sterilisation operation will be arranged for her and if she refused that operation she will have to pay a tax when the fourth child is born!

If the people are to be shaken out of their apathy, and if a fair measure of control is to be effected in the near future, some kind of legal compulsion is necessary. There can be no ethical objection to such taxation. It is only a demand on those who add to the State's heavy financial burden, to share that burden. But in a democratic country like India, a legislative measure of that kind has little chance of success unless it is made a non-party issue. For that intensive cultivation of public opinion is necessary.

It should be the duty of the medical profession, especially of the coming generations, not only to be well-informed of the why and wherefore and the techniques of family planning but also to give a correct lead to the people on this vital question.—*Reprinted from the 1959 issue of the Magazine of the Christian Mission Medical College, Vellore.*

PHYSIOLOGY AND BIOCHEMISTRY IN INDIA*

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To this learned assembly, I am sure, no apology is needed on my part for coupling the subject of physiology with biochemistry in my opening remarks. Biochemistry is closely allied to physiology. It was originally known as physiological chemistry, and is still known by that name in many institutions. It was in the 19th century that biochemistry took birth and in the 20th century it grew rapidly and has come of age. In the study of biological phenomena, chemistry and physics have been of great help, and in fact have proved of fundamental importance. Advances in these physical sciences have contributed to the advancement of all biological sciences, including physiology and medicine. Organic chemistry was founded by Frederick Whöler by his artificial synthesis of urea. It showed that organic compounds were subject to ordinary chemical laws. Colloid chemistry was founded by Thomas Graham. This is of great value to biology as it is found that many living tissues and many other substances may exist in a colloidal state. He evolved laws governing the diffusion of gases and those of osmosis and studied the dialysis of solutions through semipermeable membranes. Another great pioneer was Justus Von Leibig who was truly a biochemist and was the first to suggest the necessity of searching in the laws of chemistry for the explanation of the important phenomenon of metabolism. The studies of A. W. Von Hoffman on organic nitrogenous substances and on synthetic chemistry contributed to the advancement of physiological chemistry. The scientific basis of nutrition was established by Max Von Pettenkofer and Karl Voit. Pettenkofer's work on bile is well-known. E. F. Hoppe-Seyler made important contributions to our knowledge of the chemistry of blood, specially the spectroscopy of haemoglobin, the isolation of haemoglobin crystals and other blood pigments.

Amongst others who contributed greatly to the advancement of biochemistry in the 19th century, I may mention (a) J. F. Haller who carried out quantitative metabolic experiments and (b) H. Von. Fehling who worked on sugar in the urine. They introduced important tests which are the basis of research. Further, Helmholtz's law of conservation of energy, embodied in the first law of thermodynamics, had enormous influence in initiating a physicochemical approach to the problems of physiology and biology generally.

In the first half of the 20th century, the most notable feature in scientific research is the orientation towards biochemical investigations. Among all branches of medicine, biochemistry is the one in which most significant progress has been made. This age has been truly called the age of proteins and enzymes. We are realising more and more that by experimental research on the biochemistry of cells and microorganisms on the secretions and excretions and on the functions of organs we can obtain knowledge which would be of great value not only in our fight against disease but also in understanding vital processes occurring in living tissues and living organisms. Fortunately the number of workers in this field has increased considerably. Great advances have been made in our knowledge of the hormones, and vitamins, proteins and carbohydrates, fats and enzymes, metabolism and nutrition. Special techniques have been developed such as ultracentrifuge, the apparatus to study electrophoresis, x-ray diffraction measurements, the applications of stable and radio-active isotopes, the electron microscope which are throwing increasing light on the complexity of structure and function of living tissues.

In this connection, I may perhaps refer especially to the work of Sir F. G. Hopkins whom I had the privilege of meeting when I was a student in Cambridge (1910-1913) and worked in his laboratories. It made deep and lasting impression on my mind. He was then working on nutritive problems, which led to the discovery of vitamins. He was the first to demonstrate conclusively, that young rats failed to grow on purified nutrients but growth could be maintained if to these nutrients small quantities of milk or alcoholic extracts of milk were added. These substances in milk, which were responsible for growth, he called *accessory food factors*. They were subsequently known as vitamins. Hopkins was a pioneer in this field and he helped to establish the science of biochemistry in Great Britain on a firm basis.

I have already mentioned at the outset that physiology and biochemistry are very closely linked together. In fact, the relationship has been so intimate that in

* From the Presidential Address at the Golden Jubilee Symposium on Biology & Biochemistry of Microorganisms at the Indian Institute of Science, Bangalore, August 1959.

tracing their history they are found to be inseparable In the early part of the 20th century, as the result of the work of Hopkins and others, a time came when biochemistry established itself as an independent basic science in its own right. Some of its outstanding achievements are in the spheres of enzymes, vitamins, antibiotics and hormones.

Let us now with this background consider the position of biochemistry and physiology in India.

The study of physiology including biochemistry started in this country with the introduction of modern medical education.

The first medical school to be established in this country was in Calcutta in 1822. Similar schools were established in Bombay (1826) and in Madras (1827). The school in Calcutta was abolished in 1835 and was replaced by a medical college the same year where for the first time instruction was imparted of the type and standard prevailing in Europe at the time. The medical school in Madras was upgraded to the standard of a college in 1847 while in Bombay the school established in 1826 was abolished in 1832; and a new medical college, known as the Grant Medical College, was started in 1845. I am happy to recall that I have had the honour and privilege to serve Grant Medical College as Professor of Physiology, Dean and Principal for a period of about 21 years.

The three medical colleges in Calcutta, Bombay and Madras are the oldest in India, and were pioneers in imparting instruction in modern medicine. Biochemistry was then taught as physiological chemistry as part of physiology. Subsequently in 1860 a medical college was started in Lahore, so that by the close of the 19th century there were 4 medical colleges in the country. Their number today is about 52 and it is likely to increase in the near future. In Mysore State alone we have now 4 medical colleges, which shows how rapid this progress has been in recent years. Apart from this, the Government of India have started in New Delhi an All-India Institute of Medical Sciences where a high standard of Medical Education is being maintained. Speaking generally, biochemistry is taught in most of these colleges along with physiology as physiological chemistry, with the exception of some colleges as in Madras, Visakhapatnam, etc., where separate departments of biochemistry exist. There is an increasing tendency to treat biochemistry as an independent science, not necessarily as a handmaid of physiology, which is all to the good.

Amongst the earlier physiologists in the medical colleges in India, we have had some excellent teachers, such as McCay and Mahalanobis in Calcutta, Moorehead, Meyer and Hutchinson in Bombay, Donovan in Madras and Caleb in Lahore, who organised the teaching of physiology on sound lines and also made contributions to the science. The number of workers in this field has steadily increased in recent years and consequently there is a marked increase in the output of research work in this field. The problems which have attracted attention are those concerned with digestion, metabolism, nutrition, vitamins, blood, cardiovascular system, and nervous system. Attempts have also been made to determine normal physiological constants for the inhabitants of this country.

Much impetus was given to the study of physiology by the establishments of a separate section of physiology in the Indian Science Congress in 1936, and by the organisation of certain societies such as the Physiological Society of India. Certain medical colleges such as the Grant Medical College, Bombay, were pioneers in starting physiological societies, which have created considerable interest in the study of the subject. The Indian Council of Medical Research, the Council of Scientific and Industrial Research, and the Indian Council of Agricultural Research are also deeply interested in it and are encouraging it in a substantial way.

I would like to refer specially to the Society of Biological Chemistry which was started in 1930 with its headquarters in the Indian Institute of Science here. It was established at a time when important developments in the progress of scientific research work were taking place in the way of expansion of the existing institutions and foundation of new ones. The universities were also increasing in number and more and more biochemical research was undertaken under their auspices. The Society of Biological Chemists have served the interests of biochemistry in an admirable manner by encouraging the study and research in the subject and by establishing contacts between scientific workers in India and abroad and thus bringing about close collaboration between them. One of its important activities is to publish an annual review of biochemical and allied research in India. This has been done since 1931. It gives a very good idea of the work which is being done in this field in India and should be carefully read by all those who are interested in biochemistry in this country.

The Indian Institute of Science, where we have assembled this morning and whose Golden Jubilee is being celebrated, is the premier scientific institute in India. It is a great centre for scientific ressearch in many fields. A professorship in biochemistry was established here in 1921. It has departments of biochemistry, pharmacology and fermentation technology. I wish to congratulate the Institute as well as these departments for the great pioneering work they have been doing in the advancement of biochemistry and wish them continued success.

I may add that biochemistry is an important subject of study and research in the Indian Institute of Biochemistry and Experimental Medicine at Calcutta and the Central Drug Research Institute at Lucknow.

It is realised more and more that biochemistry is not only useful in the progress of medicine but also in the progress of agriculture and other biological sciences. So, apart from the institutes mentioned above, it is also the subject of study and research in certain other institutes such as National Chemical Laboratory, Central Food Technological Research Institute, Central Leather Research Institute, National Dairy Research Institute and others.

The actual scope of biochemical studies has been steadily increasing, so that it now comprises enzymes, vitamins, human and animal nutrition, pharmacology,

animal and human pathology, animal reproduction, dairy science, chemistry and technology of foods, fruit and vegetable preservation, soils, fertilisers, plant physiology, plant pathology, biochemistry of sanitation, genetics and microbiology.

It is also evident, that starting from modest beginnings as physiological chemistry in the 19th century, biochemistry has now reached a stage in the middle of the 20th century, when its boundaries are ever widening and the demarcation between biochemistry and biophysics is becoming more and more vague. There are definite indications that biochemistry is joining hands with biophysics in friendly collaboration which augurs well for the future of scientific progress.

As you know, we have here today a symposium on "Biology and Biochemistry of Microorganisms". This reminds me of the fact, that it was in the study of microorganisms, that organised medical research commenced in India at the close of the last century. A severe epidemic of plague had broken out and it was as the result of Haffkine's work on plague that the first medical research institute was started in Bombay in 1899. It was subsequently known as the Haffkine Institute. After that a number of other laboratories were opened, namely the King Institute at Guindy, Madras, in 1903-04; the Central Research Institute at Kasauli in 1906; the Pasteur Institute of Southern India at Coonoor in 1907; the Pasteur and Medical Research Institute at Shillong, Assam, in 1917; the School of Tropical Medicine in Calcutta in 1922; the All-India Institute of Hygiene and Public Health, Calcutta, in 1932; the Malaria Institute of India, Delhi, in 1940; Imperial Agricultural Research Institute, New Delhi, in 1935; Virus Research Centre, Poona, and others.

The work done in most of the old research institutes in the early stages was bacteriological. It was the pioneer work of Louis Pasteur and Robert Koch as well as that of Edward Jenner in Europe which was the main source of inspiration in India so that the Laboratories here had for their function the task of doing research work, mostly bacteriological in nature, which consisted in the identification and classification of microorganisms, the study of their pathogenic properties, the preparation of their cultures, preparation of prophylactic and therapeutic vaccines and sera. Routine bacteriological work was done for diagnostic purposes. Biochemistry of the microorganisms did not come into the picture very much. This is a comparatively recent development, which is to be highly welcomed. As you will see, biochemical studies of microorganisms gives us very valuable knowledge which is very useful not only in medicine but also in agriculture, industries and other fields.

NOTES AND NEWS

$8,400,000 for Health Work in S.E. Asia

Health programmes in 1961 involving a total expenditure of nearly 8,400,000 US dollars, were approved by the S.E. Asia Regional Committee of WHO during its 12th Session.

Among the resolutions passed by the Regional Committee were the following :

Eradication of smallpox—The Committee noted that the technical discussion group had unanimously agreed that smallpox was a major public health problem in most countries in the Region and that the initiation of an eradication programme should receive the highest priority among immunisation programmes undertaken.

The Committee recommended that the Regional Director pursue the problem in consultation with Regional Governments and explore the possibility of obtaining international and bilateral assistance.

Governments in the Region, where smallpox is still a major public health problem, were pressed to take urgent action towards implementing their plans for the eradication of the disease.

Technical discussions—The recommendations resulting from the technical discussion on "The Role of Immunisation in Communicable Disease Control" were that as soon as the major problem of smallpox is brought under control, mass immunisation campaigns against whooping cough, diphtheria, tetanus and typhoid fever should be started; in the meantime, immunisation programmes against the latter diseases should, wherever possible, be carried out through the existing medical and public health service.

The initiation of such measures would serve to prepare the population to receive the programme on a mass scale, when opportunities are considered suitable.

In the general discussion of the several diseases mentioned above, the group stressed the importance of the collection of essential data on the extent and pattern of these diseases by the establishment of epidemiological units; only thus can an immunisation programme be planned and undertaken in an effective and economical manner. WHO assistance with consultants and the provision of fellowships is being given towards the establishment of epidemiological units.

Typhoid was recognised to be a problem of considerable magnitude in the region. Eventual control of the disease will, of course, depend on improvements in environmental sanitation and household and personal hygiene. This, however, is a long-term measure. The group recommended that, in the meantime, immunisation against typhoid fever should be undertaken as a routine preventive.

In the control of *cholera*, it was recognised that vaccination with specific vaccine was the most widely practised prophylactic measure. The importance of initiating more studies in the field and laboratories in order to obtain more knowledge on the disease and its prevention was stressed.

The role of *BCG vaccination* in the prevention of tuberculosis in a community was discussed. In view of the marked prevalence of this disease in the countries of South-East Asia, protection with BCG vaccine as early in life as possible was recommended.

During the discussion on *poliomyelitis* the group considered that this disease was not yet a major public health problem in this region. It was, however, recommended that public health authorities should keep a constant watch over any possible changes in the inci-

dence and severity of the disease. It was considered advisable to import stocks of SALK vaccine from reliable laboratories in order to protect certain groups of people in key positions.

The participants reported that *rabies* is a problem in all the countries of the region. Ceylon hopes to carry out a mass campaign against rabies on an island-wide scale. Towards this purpose, the public health and veterinary public health services will jointly co-operate. In Thailand and Burma, such campaigns are greatly handicapped by religious feelings against the destruction of stray dogs which are, in the main, responsible for maintaining and transmitting the infection.

Virus-Destroying Bacterium

Viruses that destroy bacteria are familiar, especially the numerous strains that prey on the *Escherichia coli* organism and provide investigators with a handy tool for studying virus genetics. There also exists at least one species of bacterium able to destroy a virus, according to a report in the *Scientific American*.

The virus-destroying bacterium is a newly identified yellow organism named *Flavobacterium virurumpens* by its discoverer, Benjamin E. Volcani of the University of California Virus Laboratory. Volcani recovered the microbe from the mud of a creek crossing the Berkeley campus. The virus it destroys is the organism that causes the mosaic disease of tobacco.

The yellow bacterium dissects the tobacco-mosaic virus into an assortment of rodlets, granules, doughnut-shaped fragments and fragments of irregular shape by attacking the virus portein enzymatically. Four or five points in the virus's protein coat appear particularly susceptible to the bacterium's action.

Volcani's discovery not only points to the possible existence of other virus-destroying bacteria; it also furnishes a new means of degrading viruses to determine their structure. Hitherto physical and chemical procedures have been the chief reliance of researches seeking to break down tobacco-mosaic and other viruses for study.

Internship/Residency Training in United States Hospitals

As announced earlier in these columns the Educational Council for Foreign Medical Graduates (ECFMG) have decided that the foreign medical graduates desirous of taking up appointments as Interns, Assistant Residents and Residents in American Hospitals after January, 1960, must have passed the 'American Medical Qualification Examination'. The next examination is scheduled to be held in India (at four centres, viz. Delhi, Bombay, Calcutta and Madras) on March 16, 1960, and the last date for submitting applications to the E.C.F.M.G. is December 16, 1959. All applicants have to be approved by the Educational Council for Foreign Medical Graduates, before they are allowed to sit for the Examination.

Members of the I.M.A. who are interested in taking this examination on March 16, 1960, should obtain the application forms, etc., immediately from the Central office of the I.M.A. (Hanging Bridge, Delhi 7) and return the same duly filled in, along with the necessary photographs and photostat copies of certificates, *direct, by Air Mail*, to Dr. Ruth E. Burrows, Assistant Director, Educational Council for Foreign Medical Graduates, Orrington Hotel, 1710 Orrington Avenue, Evanston, Illinois, U.S.A. to reach her before December, 16, 1959.

Rockefeller Grants to India

Indian Institutions and individuals received nearly $140,000 as grants and fellowships from the Rockefeller Foundation during the third quarter of 1959. The Foundation on 2-11-59 named scientific, educational, medical and agricultural organisations in 33 countries which were awarded grants totalling $1,556,082 for individuals from 30 countries who were awarded fellowships which became tenable during the period.

The Foundation said that grants awarded to further research and study in the medical and natural sciences amounted to $546,135; agricultural sciences $567,360; social sciences, $112,975 and humanitarian projects, $166,912.

I.C.M.R. Proposals for Medical Research

The Indian Council of Medical Research invites applications for grants-in-aid for carrying out research during 1960-61 (from 1st April, 1960 to 31st March, 1961) on fundamental and applied aspects of any of the problems listed below in Institutions where adequate laboratory and other necessary facilities for conducting research are available :

A. *Communicable Diseases* :
1. Tuberculosis
2. Cholera
3. Leprosy
4. Infantile diarrhoea
5. Typhoid
6. Filariasis
7. Poliomyelitis
8. Diphtheria
9. Smallpox

A.1 *Virus Diseases* :
1. Respiratory viruses
2. Enteric viruses

B.
1. Malnutrition
2. Venereal diseases
3. Helminthic infections
4. Amoebiasis
5. Dental health
6. Cardiovascular diseases
7. Metabolic Disorders

C. Environmental Sanitation

D. Industrial Health

E. Trials of Indigenous Drugs.

Applications in triplicate on the prescribed application form, with 120 copies of a note giving details of lines of work proposed to be undertaken on the enquiry, should be submitted through proper channel to the Director, Indian Council of Medical Research, New Delhi, so as to reach him not later than *20th November, 1959*. Application forms can be supplied on request by the Director, Indian Council of Medical Research.

N.B.—It may please be noted that schemes on the subjects mentioned against 'A' to 'E' in the list referred to above, received from research workers for financial support in 1959-60 and which were recommended by the Scientific Advisory Board of the Council for acceptance,

would be reconsidered at the annual meetings of the Council in December, 1959, and that it is not necessary for those workers to submit those schemes over again for grant of funds in 1960-61.

All-India Ophthalmological Conference, 1960, Agra

The XXI session of the All-India Ophthalmological Conference will be held at Agra in the first week of February, 1960. More then 200 delegates will be attending the conference from all over India, and some distinguished ophthialmologists will also be coming from abroad. A Symposium on "Hypertensive Retinopathy" will be held during the session.

New Breakthrough in Cardiac Surgery

A woman fitted with a rabot heart valve made of plastic nearly a year ago, is "progressing satisfactorily", an American surgeon reported on 24-10-59.

Dr. William H. Muller Jr., a University of Virginia surgeon, gave details of this first complete replacement of a human heart valve—heralding a possible new breakthrough in cardiac surgery—to the American Heart Association meeting at Philadelphia.

He said the plastic valve was a direct replacement for the valve of the aorta.

Dr. Muller told the annual scientific meeting of the Heart Association that the plastic valve was designed to correct aortic valve insufficiency.

Dogs Carriers of Human Epidemic Strains

Dogs can be carriers of human epidemic strains of antibiotic-resistant staphylococci, it was revealed in a report presented to the American Public Health Association's 87th annual meeting on 2-10-59.

The report was made by a veterinary surgeon from India and two co-workers from Kansas. They are Dr. P. S. Rajulu, Principal of the Veterinary Medical College, Tirpuati, Andhra Pradesh, who worked on the study as a graduate student at Kansas State University and two professors of bacteriology at the Kansas Agricultural Experiment Station, Dr. T. H. Lord and Mr. V. D. Foltz, who presented the report.

New Disease Identified as Bilharziasis

Bilharziasis, a water-borne disease prevalent in Egypt, Japan and Brazil but unknown in India till now, has been found in an endemic form in Gimvi, a village near Ratnagiri, by Dr. R. K. Gadgil, Head of the Department of Pathology in the Grant Medical College, Bombay.

Dr. Gadgil said that an Indian snail has been found to be a new intermediate host. The snail is *Ferissia tenius*. Till now, only two types of snail, Bulinus and Physopsis, have been identified as intermediate hosts.

It took the Bombay pathologist seven years to identify the disease. He first noticed it in Gimvi in 1952. He established the life-cycle of the parasite by using the larvae emitted by infected *Ferissia tenius*.

Gift of Mobile Medical Van

A complete mobile medical and surgical unit donated by a lawyer and philanthropist of Philadelphia, U.S.A. Mr. Graham French who visited Bombay last June, was presented to the Bombay Mothers and Children Welfare Society, in the compound of the World Maternity Home, Bombay, on 21-10-59. The mobile unit, which will serve 3,000 villagers in the Khed area near Poona, will concentrate on prenatal and postnatal work.

This specially designed van has been built to meet the particular needs of the villages of Chakan, Kadus, Chas and Path and the roundabout villages on the Poona-Nasik road.

Efficacy of B.C.G.

Dr. C. R. Naidu, Director of Public Health, inaugurated the BCG Vaccination campaign at Kurnool, at a function held at the Clinical Lecture Hall, Kurnool Medical College, before a gathering of medical men and the public.

Dr. Naidu said that though tuberculosis was a scourge, it was now curable by domiciliary treatment. Better nutrition and preventive steps such as BCG vaccination, could keep tuberculosis away. He added that BCG was the only known immunity. Two rounds of mass BCG vaccination had been completed in Telengana and in Andhra the first round will be over shortly.

Dr. Gurumurthy Memorial Prize Essay Competition

The above competition has been announced by the Andhra State Branch, I.M.A. The subject of the essay is Hyperpiesia (Hypertension) in General Practice with special reference to Ætiological Factors and Treatment. Only members of the A. P. branch are eligible to compete for the Gurumurthy gold medal. Further information may be obtained from the Hony. State Secretary, Andhra Pradesh State Branch, Sultan Bazar, I.M.A. Building, Hyderabad, A.P.

Prize Competition Essay

A Prize Competition Essay has been jointly sponsored by the *Bombay Hospital Journal*, Bombay and Messrs. Lederle Laboratories (India) Private Ltd. It is open to all medical practitioners (registered under the Indian Medical Council). The subject of the essay is "How to be a successful general practitioner". The first prize is worth Rs. 1,000/-, the second prize Rs. 750/- besides other offers. Further particulars may be obtained from the Editor *The Bombay Hospital Journal*, 19 Marine Lines, Bombay 1.

Silver Jubilee Celebration of N. M. Wadia Charitable Hospital, Sholapur

The Silver Jubilee Celebration of the N. M. Wadia Charitable Hospital, Sholapur concluded on October 18, 1959. The celebration was inaugurated by Dr. A. V. Baliga of Bombay at the hospital premises.

The N. M. Wadia Charitable Hospital owes its present status to the efforts of Dr. V. G. Vaishampayan

who started a dispensary in 1933. The dispensary was later converted into a hospital from a donation of Rs. 10,000/- from the N. M. Wadia Charity Trust, Bombay. The hospital with 24 beds was declared open in 1934. It gradually expanded and now has 15 sections with 150 beds. The hospital has a small wing for tuberculous patients and also a blood bank, and a chest surgery centre. A batch of 15 graduate and post-graduate doctors all of whom are members of Indian Medical Association work on its staff with great co-operation.

On this occasion the management has collected a donation of one lac and thirty thousand rupees from its sympathisers and public at large, to be able to expand the hospital further. It is proposed to have more beds, a separate T.B. hospital, an occupational therapy centre, and an up-to-date pathological laboratory.

New Hospital at Malad

A 360-bed hospital is proposed to be opened at Malad by the Kailas Seva Sadan Trust from a donation of Rs. 2 lakhs made by the Mahadevi Piramal Charitable Trust of Morarji Gokuldas Mills, Bombay. The hospital will be known as the Piramal Hospital.

This will be the biggest hospital in Bombay to be run by a charitable trust. It will be the only charitable hospital in the city to have brain, heart, chest, urinary surgery and cancer departments.

Bombay's Deputy Minister for Public Health, Dr. N. N. Kailas, who is the Chairman of the Kailas Seva Sadan Trust, said that the hospital buildings and quarters for the staff were expected to cost Rs. 23 lakhs and the equipment another Rs. 7 lakhs. He said the hospital buildings would be ready for occupation by March, 1961.

The proposed hospital would be located near the S. K. Patil Arogyadham.

Eradication of Smallpox

The Andhra Pradesh Government are contemplating the launching of a smallpox eradication scheme during the third five-year plan period. The Central Expert Committee, constituted by the Government of India, after consultations with the State Expert Committees, has recommended such a programme.

As a first step, a pilot project was launched in the Vijayawada municipal area, and it was subsequently extended to the Visakhapatnam, Masulipatam, Khammam, Karimnagar, Hyderabad, Guntur, Mahbubnagar, Medak, Nizamabad, and Nalgonda districts. The idea is to cover nearly 60,000 persons, and the unit is expected to complete the work within a month.

Madras Medical College Expansion Scheme

The Madras Medical College will have additional buildings by the middle of next year to accommodate the Dental Wing, the Chemistry Block and the Physiology Block. The new buildings are expected to cost nearly Rs. 40 lakhs.

The Physiology-cum-Pharmacology Block, which is being constructed at a cost of Rs. 20 lakhs (inclusive of laboratory fittings) will be completed in another three months. The Physiology Block is at present located at the Anatomy Block for want of accommodation.

The Chemistry Block, which will be a three-storeyed building, is being constructed at a cost of Rs. 9·8 lakhs. At present the Chemistry Block is located in the Pathology Block. Chemistry, Biology and Physics are among the subjects taught here.

The new accommodation for the Dental Wing under construction at an estimated cost of Rs. 9·2 lakhs will be a new two-storeyed building.

After the introduction of the Pre-Professional Medical Course and other courses like B.D.S. the authorities of the Medical College have been experiencing difficulty in providing adequate laboratory and other facilities for students undergoing the courses. The new blocks, when completed, are expected to provide sufficient accommodation for theoretical studies and practical experiments in an atmosphere of comfort.

Health Schemes in Third Plan

The Union Ministry of Health is understood to have prepared tentative proposals, costing about Rs. 415 crores, for inclusion in the Third Five-Year Plan with particular emphasis on the total eradication of communicable diseases and the supply of drinking water.

Some of the other items proposed to be given high priority include medical education, teachers' training, the establishment of rural and urban health centres and family planning.

A comprehensive scheme for the eradication of malaria is already under execution. Besides giving it priority in the Third Plan, a vigorous campaign is proposed to be launched for wiping out the two other major diseases, namely, cholera and smallpox by 1965. Details of the campaign are being worked out in consultation with the State Governments.

The Planning Commission's working group for formulating health schemes in the Third Plan has suggested that a high priority should be given to water supply and sanitation schemes. A large financial provision is sought for implementing the programme.

Special attention will be given for checking the deteriorating standard of teaching in medical colleges owing to the paucity of trained teachers.

A detailed survey of the existing teaching facilities, available in the various institutions, conducted recently, showed that there was a total strength of about 2,000 teachers to all the medical colleges. Hence the emphasis may have to be more on the expansion of the existing colleges than on starting new ones.

The Family Planning Board, which is at present engaged in formulating concrete proposals for family planning, is reported to be of the view that family planning services should be made available in the Third Plan at 36,000 medical and health institutions in India.

With a view to achieving this target, it is proposed to train the staff working in these institutions.

500-Bed Hospital in Ahmedabad

The Ahmedabad Municipal Corporation will shortly open a 500-bed hospital as an adjunct to the tuberculosis

dispensary at Ahmedabad. The incidence of tuberculosis in Ahmedabad is one in 40.

The announcement was made by the Mayor, Mr. Chinubhai Chimanial at a function where Mr. Amritlal Hargovindas, a well-known industrilist, declared open a 34-bed tuberculosis ward at the dispensary.

Mr. Hargovindas praised the Katpitiya Mahajan, an organisation of timber merchants, whose gift made the new ward possible.

Pfizer Patent on Tetracycline Recognised

Validity of Pfizer's product patent on tetracycline has been recognised in a Court-approved settlement in England.

In January of this year Pfizer brought legal action in England against Ward Casson Limited for infringement of this patent. A settlement of this action has now been arranged and this was approved by Mr. Justice Lloyd-Jacob in the High Court on 31st July, 1959.

Tetracycline is an antibiotic extensively used in a wide variety of infections. Pfizer's trade name for the product is Tetracyn.

Health Conditions in India During July

Births and deaths registered in towns with a population of 30,000 and over during June, 1959, were of the order of 23 and 11 per thousand of population respectively. These figures are the same as in the month of May, 1959.

According to available data, the country remained free from plague. Cholera and smallpox, however, continued to be reported from some parts of the country.

Cholera: Based on preliminary data for the week ended July 25, 1959, mild incidence was reported from Patna and Palamau districts in Bihar State; Ahmednagar and Nagpur districts in Bombay State; Guna district in Madhya Pradesh; Mandya district in Mysore State; Ganjam district in Orissa; and Calcutta city.

Smallpox: Mild incidence was reported from the districts of Srikakulam, East Godavari, West Godavari, Krishna, Nellore, Kurnool, Anantapur, Guntur and Hyderabad in Andhra Pradesh; Nagpur, Greater Bombay, North Satara and Poona in Bombay State; Raisen district in Madhya Pradesh, Ferozepur district in Punjab; Kotah and Tonk districts in Rajasthan; the districts of Coimbatore, the Nilgiris, Madurai, Salem, Madras city, Chingleput, Tiruchirapalli and Kanya Kumari districts in Madras State; Tumkur, Mandya, South Kanara, Dharwar, Belgaum, Chikmagalur, Bellary and Bijapur districts in Mysore State; Puri, Sambalpur, Dhenkanal and Mayurbhanj districts in Orissa, and Pondicherry.

Gastroenteritis: During the week ended July 18, 1959, 131 cases with 25 deaths were reported from Uttar Pradesh as against 160 cases and 40 deaths during week ended July 11, 1959, and 215 cases and 45 deaths during week ended July 4, 1959.

Kyasanur forest disease: Three attacks were reported from Shimoga district in Mysore State during the fortnight ended July 18, 1959.

Influenza: Reports of sporadic cases were received from Assam, Bombay and Mysore States during the month under review.

Quarantine Restrictions Withdrawn

Information has been received by the Director-General of Health Services that the Thailand health authorities have withdrawn quarantine restrictions on account of smallpox against passengers from Calcutta port and airport with effect from September 2, 1959.

CORRESPONDENCE

The Editor is not responsible for the views expressed by correspondents

Parenteral Diethylcarbamazine in Tropical Eosinophilia

SIR,—We have read with interest the article of Misra *et al* which appeared in your Journal of 16th March, 1959 (p. 232) and the letter by Sheth and Shah in the July 16, 1959 issue (p. 67).

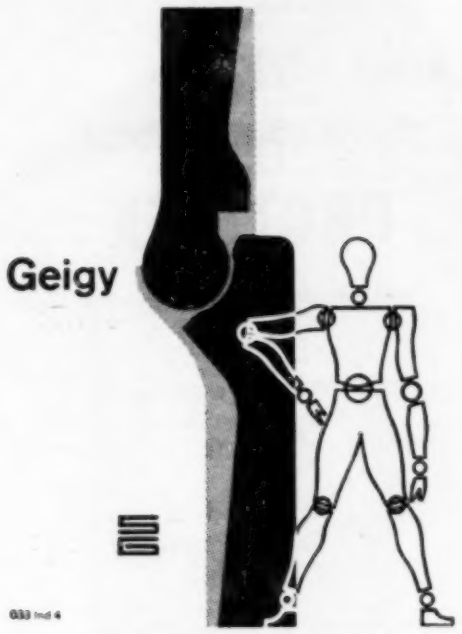
Several authors have now reported the efficacy of diethylcarbamazine in treatment of tropical eosinophilia and we have also confirmed the same in a study of 26 cases. We took up an experimental study to elucidate the mechanism of action of diethylcarbamazine in order to throw some light on the aetiology of this disease. Two theories are usually postulated for this condition—(a) the allergic theory and (b) the infection or infestation theory (Chaudhury *et al*, 1954). Both the theories were tested.

It was found that unicarbazan even in doses approximating the LD-50, that is 400 mg/kg body weight, did not modify anaphylactic shock in either guinea-pigs or rats, sensitised and challenged by horse-serum or fresh egg-white solution. Further, there was no inhibition of the anaphylactic contraction of the isolated smooth muscles of the guinea-pig and the rat in a concentration of 10^{-3}.

The parenteral preparation (unicarbazan) antagonised the action of histamine on the isolated guinea-pig ileum, but not on the blood pressure of the atropinised cat under urethane anaesthesia. In the latter preparation, injections of unicarbazan caused a rise in blood pressure lasting for several minutes. The powerful antagonistic action of unicarbazan on the guinea-pig ileum is partly due to its antihistaminic constituent—'Diphenhydramine', and partly due to diethylcarbamazine, itself, which in a concentration of 10^{-4}, reduced the histamine effect by 50 per cent.

Unicarbazan did not reduce the eosinophil response in rats and more particularly in the guinea-pigs, after injections of histamine, the histamine-liberator compound 48/80, or after nonfatal anaphylactic shock. Thus it seems this drug does not specifically inhibit the eosinophil response.

When guinea-pigs were fed saline extracts of stool containing roundworm ova, or roundworm larvae, a tenfold rise in the eosinophil count was demonstrated from 3rd to 7th day. Unicarbazan (in a diosage of 100



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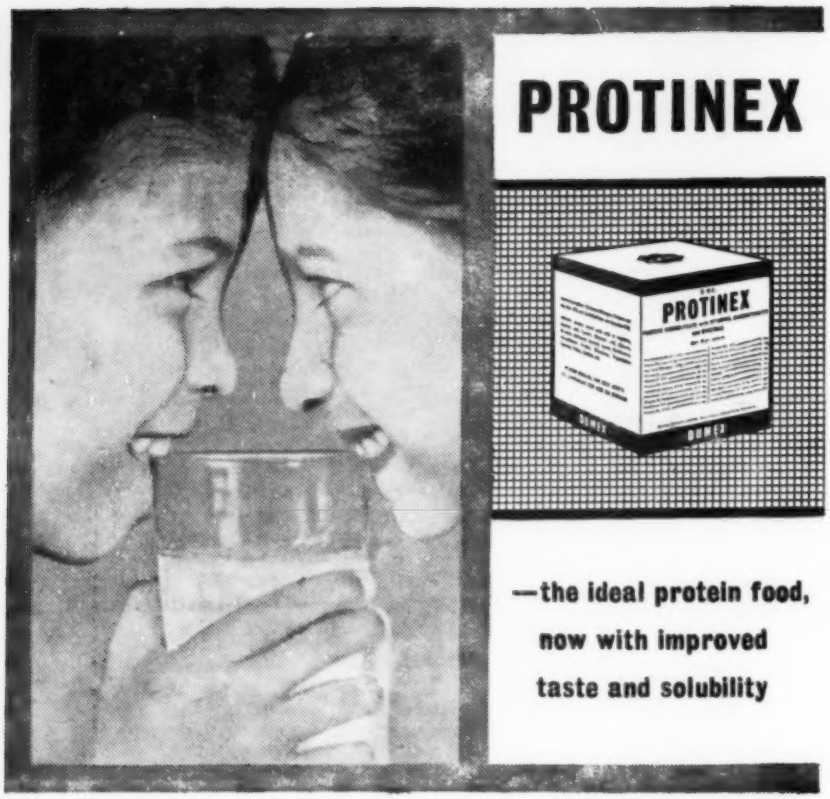

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mg/kg, 2 daily injections) completely inhibited this rise in eosinophil count. Freshly passed round worm ova is not infective, and it is possible that the stool contains a factor probably elaborated by the parasite which causes a rise in the eosinophil count. Ascaris extracts are known to produce anaphylactoid symptoms (Rocha e Silva, 1955). The inhibitory effect of unicarbazan thus may be due to its anthelmintic effect and as well to its antagonising effect on the elaborated toxic factors responsible for causing eosinophilia.

Parasitic infestations are quite common in tropical eosinophilia. Chaudhury *et al* (*loc. cit.*) have already listed the various parasites that have been found in cases of tropical eosinophilia. Recently Danaraj (1959) has shown a high incidence of filarial complement fixation test in such patients. The difficulty in accepting an 'Infestation theory' is the fact that many patients with such infestations remain free from symptoms, characteristic of tropical eosinophilia. Our theory is a modified form of the infestation theory reconciling this anomaly.

It was demonstrated by Jha (1957) that in distinction to the normal populace, patients of tropical eosinophilia exhibit a hypersensitivity to histamine, along with a raised histamine content of the blood. During the last decade, a large number of substances including helminthic parasite extracts have been shown to release histamine (Paton, 1957). It is suggested that increased sensitivity to histamine is the prime factor in tropical eosinophilia, histamine release being conditioned by a variety of factors including parasitic infection. The efficacy of diethylcarbamazine is not related to its anti-anaphylactic property but is related to its anthelmintic action.

What causes the primary histamine hypersensitivity is at present obscure. We are etc.

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Darbhanga Medical College,
Laheriasarai, Bihar.

R. K. Sanval,
B. P. Mishra,
B. B. Sinha.

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Medical Licentiates

Sir,—During foreign rule, it was necessary for the rulers to please the ruled by some humanitarian activities, one of which was medical relief at a lesser cost. With this aim in view some medical schools were started in the country and medical licentiates produced.

The All-India Medical Licentiates Association, had in its objectives, the abolition of 'caste system' in the medical profession. The Indian Medical Association too, played its part in it. The schools are now closed, the foreign regime having been changed in the meantime to national regime, everybody expected that this class system could no longer exist. But alas! that was not to be. Except a very few exceptions who rose up higher and higher by their merits and personal efforts, the class remained as it was along with a distinct deplorable difference at the present time, namely exclusion in appointments in all sectors. Medical licentiates are now untouchables in services either in Government concerns like Life Insurance Corporation, Health centres, Government institutions etc. or in private enterprises like factories, tea gardens and collieries, etc. Again, while the Government has declared that the minimum basic qualification of a medical man is M.B.B.S., the universities in the country had either completely closed the doors or modified the rules in such a way that the unfortunate licentiates cannot now upgrade themselves through Condensed M.B.B.S. course or D.T.M. course, that were open to them for some years. Now let us imagine the fate and career of a medical licentiate just passed out in 1955. He will not be able to upgrade himself to acquire minimum basic qualification to get a Government job nor will he be able to secure any job anywhere in the country. The only way open to him is to try his luck in private practice without any further training. If luck does not favour him, what will be his fate except starvation with his family?

We appeal to the members of the organised medical profession in the country as well as to our national leaders at the helm of administration to have a soft corner in their heart of hearts, for these unfortunate licentiates. Why should they be the worst victims for no fault of theirs? We appeal most humbly and sincerely for an early solution of this problem and thereby save the licentiates from the sufferings that stare at them. I am etc.

Dhakuria. A. C. Sen Gupta.

REVIEWS

Surgery of the Head and Neck—The Year Book Publishers, Inc., Chicago, U.S.A. Size 6¼"×9", pp. 319, price $9.75.

This is one of the Handbook series which is being published by the Year Book Publishers from Chicago. In conformity with the high standard set up by other similar volumes of the series, such as, Thorax, Abdomen, etc., this book also is highly informative and illustrative. The subject of head and neck surgery is complicated and is usually indifferently understood by general surgeons. It is expected that this book will fulfil a very important need.

Urology in Outline—Published by E. & S. Livingstone Ltd., Edinburgh. Size 5½"×8½", pp. vii plus 176, price 27s. 6d.

This book is a very interesting one. The subject of urology including the clinical features of diseases, diagnostic studies, pathological features and surgical

management have been presented mostly in the form of diagrams. The diagrams are line drawings and there is some advantage in this type of presentation in contrast to the usual text-book descriptions. The book is not an exhaustive outline of the subject but presents most of the essential features. For quick revision of the subject of urology, particularly for the post-graduates, this book will be invaluable.

Childbirth without Pain—By Dr. Pierre Vellay and others. Translated from the French by Denise Lloyd, Publishers : Hutchinson of London with George Allen and Unwin. Size 9"×6", pp. 216, price 35s. net.

This book is meant for the laity, viz., the expecting parents. It gives a very good account of the natural phenomena associated with reproduction. Beginning with fertilisation of the ovum and ending with the completion of labour the description is elaborate. The language of narration is such as will be easily understood by a lay person of average literary knowledge. The system described is meant to be easily followed and to be practicable with guidance by experts specially trained.

There are several elaborate personal accounts given by patients who have followed and been benefited by the system. There are a few photographs of actual confinements where the physiognomy of the patient would show that she does not present the picture of one afflicted with pain or panic.

So far so good. The question naturally rises in the enquiring mind where is the usefulness of this book in so far as institutional obstetrics is concerned. In this country as in many others throughout the world where a big percentage of confinements take place in institutions, the pain borne by patients during normal confinements does not cause anxiety. The relief from pain, however, as obtained through skilful instructions given by persons especially trained for the purpose to co-operating and intelligent patients, though desirable does not appear to be economically possible. The system is likely to be appreciated and utilised by the few who may be confined in nursing homes where special fees have to be paid for the expert services received.

Furthermore, the details of the system as depicted in the book do not give sufficient material to enable one to pass an opinion about the scientific elucidation of whatever result might have been achieved.

The book is however a very pleasant reading treatise. The personal accounts are interesting. The get-up is good. The photographs are well reproduced.

Pathology of Tumours of the Nervous System—By Dorothy S. Russell and L. J. Rubinstein. Edward Arnold Ltd., London, 1959. 7¼"×10", pp. 318, price 70s. nett.

This monograph contains 14 chapters, the last chapter dealing with tissue culture in relation to tumours of the nervous system. In the pathogenesis of tumours of the nervous system, some special features have to be taken into account, such as the high incidence of tumours in childhood, the possible part played by embryonic rest and family history, especially in tuberose sclerosis and von Recklinghausen's disease, and the element of maldevelopment in the teratomas, dermoid and epidermoid cysts. The role of trauma in the pathogenesis of meningiomas is not negligible. The meningiomas are most commonly found in the middle decades of life with predominance in women. Calicified particles known as psammoma bodies and whorled pattern are visible on the cut surfaces of the tumour. In the malignant form the picture is that of a spindle-celled sarcoma. The tumours of the reticular tissue include Hodgkin's disease, lymphoma, sarcoma and leukaemias. They are more numerous in the grey matter. In Hodgkin's disease the spinal form shows neurological symptoms of spinal compression and transverse myelitis. Cavernous angiomas have predilection for the neighbourhood of the Rolandic fissure giving rise to Jacksonian epilepsy. In Sturge-Weber-Dimitri's disease there is an extensive capillary-venous malformation affecting one cerebral hemisphere. It is evident as a tram-line type of calcification in the x-ray picture with port-wine stain in the trigeminal region with contralateral hemiparesis and Jacksonian seizures. The neuroglial tumours are known as gliomas and these constitute the most important intracranial tumours The males are more often affected. The tumours in childhood are subtentorial while in adults these are supratentorial. The behaviour of these tumours is unpredictable. The tumours of the neurone series are the medulloblastomas. The disturbances seen in pubertas praecox are of hypothalamic origin and not due to pineal tumours. Retinoblastoma vies in malignancy with the cerebellar medulloblastoma. In tuberose sclerosis there are retinal astrocytomas found in mentally deficient children with adenoma sebaceum and epileptic fits. In the secondary dural involvement the most frequent primary sources of carcinoma are the lungs, prostate, stomach and breast. Pressure cone is due to downward thrust of cerebellar tonsils through the foramen magnum and the fatal result is due to the compression of the medulla oblongata. Phaeochromocytomas mostly found in adrenal glands cause persistent hypertension because of the production of adrenaline and noradrenaline. In tissue culture the vigour of growth is an index of malignancy in general. Tissue culture is essentially a supplementary and research method. There are 286 illustrations in the book and exhaustive references at the end of each chapter. The authors have taken great pains in preparing this monumental work on the difficult subject of tumours of the nervous system. This monograph will serve as a source book for the guidance of research workers in the field.

Reconstructive and Reparative Surgery—By Hans May. Published by F. A. Davis Company, Philadelphia, Sole Agents in India, Kothari Book Depot., Bombay, Pp. 1115, with 1030 illustrations, 55 in colour, size 10"×6¼", price Rs. 135/-.

This book on plastic surgery gives a fair amount of information and deals with some subjects of construction of oesophagus (total jejunal oesophagus) that are

now in the domain of thoracic surgery. These chapters could be utilised in a better way by giving more details of the main plastic problems. From omissions like use of Kilner's gag in cleft palate, etc., one wonders whether political considerations are coming into text books of medicine and surgery.

Hernia—By Sir Heneage Ogilvie, Published by Edward Arnold (Publishers) Ltd., London, W.1., Sole Agents in India, Orient Longmans (Private) Ltd., Calcutta 13, size 8½"×5½", Pp. 135, price 28s. nett.

The book gives a detailed description of anatomy, comparative anatomy and surgery of the disorder named hernia. The treatment and the diagrams are so excellent that even an undergraduate can grasp the whole subject. Monographs like this from master minds such as Sir Heneage are real treasures.

Eisenstoffwechsel : Beiträge zur Forschung und Klinik, Bearbeitet von zahlreichen Fachgelehrten—Herausgegeben von Prof. Dr. W. Keiderling, Freiburg/Br. 1959 XIV, 298 Seiten, 138 zum Teil mehrfarbige Abbildungen, Gr.-3°, (Georg Thieme Verlag, Stuttgart), Ganzleinen DM 48.

Iron Metabolism, contributions to research and clinic—Edited by W. Keiderling, Freiburg/Br., with 30 collaborators. Pp. 298, 138 partly multicoloured illustrations (in German). Price DM 48.

The occasion for bringing out this remarkable monograph was the 60th birthday anniversary of Prof. L. Heilmeyer whose name is closely linked with our knowledge of iron metabolism that has made such remarkable progress during the last 25 years. Thirty outstanding experts, not only from Germany but also from France, the U.S.A., Switzerland, Sweden and Hungary, contributed chapters in German, French and English on the methods of iron research, its physiology, pathology, clinic and toxicology.

The methods included cover histochemistry, the estimation of ferritin and haemosiderin, electron-microscopic studies and ferrokinetics. The second main chapter deals with the function of the various body iron compounds, such as myoglobin iron, cell haemins, depot iron ferritin, haemosiderin and plasma iron. Then follow some 50 pages on various aspects of the physiology of iron transport, resorption, conservation, elimination and regulation of the iron household. One hundred pages are devoted to the most important pathological conditions, such as the various kinds of anaemias and polycythaemia, infections and neoplasmas, liver diseases, haemosiderosis and haemochromatosis; particularly interesting seems to this reviewer Waldenström's chapter on sideropenia and Björkman's on anaemia refractoria sideroblastica. Two articles on iron therapy and acute oral iron intoxication conclude this well edited and fairly well balanced volume. The beautiful paper and clear print, the large number of excellent microphotos, some of which are in colour, substantially enhance the pleasure of handling this birthday present which is worth of the meritorious sexagenarian in whose honour it was published.

Die Abnützungserkrankungen der Sehnen und ihre Therapie—Von Doz. Dr. H. Schneider, Innsbruck. Mit einem Vorwort von Prof. Dr. G. Hohmann, München, 1959. VIII, 202 Seiten, 52 Abbildungen, Gr.-8°, (Georg Thieme Verlag, Stuttgart), kartoniert DM 36.

Diseases of Wear and Tear of the Tendons and their Treatment—By Doz. Dr. H. Schneider, director, Institute of Physical Therapy, Tirolese Panel Institution, Innsbruck. Preface by Prof. Dr. G. Hohmann, Munich. Pp. 202, 52 illustrations (in German). Price DM 36.

This slum volume is packed with information based on the author's wide experience as an anatomist as well as a practical physiotherapist. The starting point is the generally overlooked fact, established by Weidenreich in 1922, that the tendons are not inserted in the periosteum but in an area free from it. Accordingly, the first 24 pages deal with anatomy and physiology of tendons and particularly with structure, function and auxiliary apparatuses in the insertion zones. This is followed by a discussion of fatty degeneration and necrosis of and calcium deposits in tendinous tissue. The next sections deal with tendon ruptures in the upper and lower extremities; regenerative transformations in the tendons; de- and regenerative changes in the insertion area. A longer chapter is devoted to aetiology and pathogenesis of tendon diseases caused by wear and tear. Symptomatology, nomenclature, frequency and the relation of tendopathies to sport follow next. Some 65 pages, one third of the book, are devoted to a systematic description of therapy, physical, medical and surgical; prevention, particularly of tendopathies of occupational origin; finally, regional therapy. At the end of every chapter a fair amount of references in many languages are listed. A good subject index concludes this stimulating and well illustrated book which should prove very useful not only for orthopaedists and physiotherapists who will find it thought-provoking and stimulating but also for physicians who have to advise the host of sufferers from pain in shoulder, arms and legs who still are frequently lumped together and maltreated as rheumatism and fibrositis.

OBITUARIES

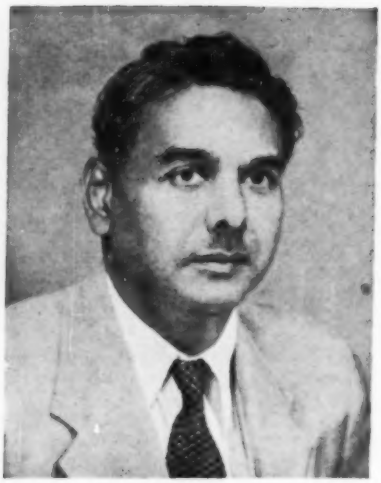
Dr. Ganapati Panja

Dr. Ganapati Panja was born in July, 1891, in a village in the district of Burdwan (West Bengal). He lost his parents at the age of seven and had therefore to struggle hard for the prosecution of his studies. He was a scholar throughout his student career. At the Medical College he was the recipient of a number of gold medals for proficiency in various subjects and obtained the F. C. Chatterjee scholarship in morbid histology and also the best man's scholarship.

He qualified as an M.B. in 1919 and served on the house staff of the Medical College Hospital for some time. He joined the Calcutta School of Tropical Medicine in 1921 as an Assistant Professor of Bacteriology. Here he came in close contact with Lt. Col. H. W.

DR. G. PANJA

DR. A. NARAYANA MENON



DR. B. A. RAJARATNAM

Acton which soon paved his career as a dermatologist of all-India fame.

Dr. Panja received a Rockefeller Fellowship in 1932 for prosecuting studies in London, passed the D. Bact. examination of the London School of Tropical Medicine and worked in the Edinburgh Royal Infirmary and various dermatological clinics in Europe. He was the receipient of the Coates Medal of the Calcutta University in 1947. He became Professor of Bacteriology and Pathology in the School of Tropical Medicine, retiring after years of fruitful service in 1948. Dr. Panja contributed a large number or original papers to medical journals both inland and foreign. He earned recognition by his original work on the Malassezia of dandruff, pityriasis versicolor, and for introducing a method for the isolation of vibrios from cholera stool. The D.E.C. medium of Panja and Ghosh was introduced as a highly successful medium for cultivation of intestinal pathogens. Dr. Panja also threw new light on the aetiology of tropical phagadaenic ulcer.

Dr. Panja presided over the medical and veterinary section of the Indian Science Congress in 1947. He was a vice-president of the Indian Medical Association, Calcutta Branch in 1949-50 and was connected with the British Medical Association as a member of the Dermatological group.

Dr. Panja was a Founder Trustee of the Indian Institute of Dermatological Research and the editor of the Indian Journal of Dermatology. His association with our journal was long and intimate and the services rendered by him were well appreciated.

Dr. Panja breathed his last on September 7, 1959.

Dr. A. N. Menon

Dr. Ambat Narayana Menon, M.B.B.S., well-known medical practitioner and prominent citizen of Madurai, passed away on the 21st of September 1959. He was the first secretary (for 7 years continuously) and one of the founder members of the Madurai Branch, I.M.A. He edited for 2 years a journal of the Madurai Medical Association. He was a vice-chairman of the reception committee of the All-India Medical Conference at Madura in 1946.

Born in 1894 at Chittur, Kerala State, he was educated at Chittur, Kozhikode and Madras, graduating in Medicine in 1920 from the Madras Medical College. He won the Dr. Nair Gold Medal for proficiency in Surgery. After serving as House Surgeon and Temporary Assistant Surgeon in the Presidency Hospitals, Government Maternity and Government General Hospitals from 1920-22, he set up practice in Madurai, which grew rapidly and attracted a wide clientele. He served as hony. venereologist, Government Headquarters Hospitals, Madurai, from 1930-39. He was connected with almost all of the social service organisations of the city of Madurai.

Dr. B. A. Rajaratnam

Dr. B. A. Rajaratnam was born on 31st August 1899. He started general practice at Salem in 1923 and was in active life till his sudden death on 17th July 1959. During these 35 years of successful practice, he won the admiration, respect, affection and confidence of all his patients.

Besides his profession, he was the Chairman of the Salem Municipal Council from 1935 to 1938, Member of the Senate, University of Madras, Member of the District Board, Salem, President, Y.M.C.A., Salem, Founder Member, District Club, Salem, and Mason of 26 years standing.

A very active member of the Indian Medical Association, he was President of the Salem Branch of I.M.A. in 1948-49 and was President of the Building Committee of Salem Branch of I.M.A. till his death.

In him the country has lost a worker of outstanding ability, the medical profession a genial, courteous and sincere colleague and the Indian Medical Association an ardent member.

SUPPLEMENT
Journal of the Indian Medical Association

VOL. 33 No. 10 CALCUTTA NOVEMBER 16, 1959

ASSOCIATION NOTES

I.M.A. CENTRAL COUNCIL *-Proceedings of the 79th Ordinary Meeting of the Central Council of the I.M.A. held at Cuttack on 28th December, 1958.*

MEMBERS PRESENT : 1. Dr. C. O. Karunakaran (Trivandrum)—President (in the Chair); 2. Dr. V. P. Vasudevan (Coimbatore)—Vice-President; 3. Dr. Ved Prakash (New Delhi)—Hony. Joint Secretary; 4. Dr. P. K. Guha (Calcutta)—Hony. Editor, Journal, I.M.A.; 5. Dr. S. C. Sen (New Delhi); 6. Dr. H. N. Shivapuri (Lucknow); 7. Dr. A. D. Mukharji (Calcutta); 8. Dr. C. S. Thakar (Bombay); 9. Dr. Arvind Shah (Bombay); 10. Dr. B. P. Tribedi (Calcutta); 11. Dr. B. L. Kapur (Ludhiana); 12. Dr. S. S. Rao (Cochin); 13. Dr. G. S. Kulkarni (Gadak); 14. Dr. M. D. Panchmia (Bombay West Suburban Branch); 15. Dr. J. J. Merchant (Bombay); 16. Dr. C. L. Amin (Bombay); 17. Dr. S. C. Sheth (Bombay); 18. Dr. M. A. Panwala (Bombay); 19. Dr. T. N. Ghosh (New Delhi); 20. Dr. S. N. Acharya (Puri); 21. Dr. J. P. Ganguli (Delhi); 22. Dr. R. K. Chakravarty (Calcutta); 23. Dr. V. P. Khare (Jabalpur); 24. Dr. S. V. Upadhye (Dharwar); 25. Dr. S. S. Ajgaonkar (Bombay); 26. Dr. M. Vasudeva Rao (Hyderabad); 27. Dr. B. K. Modak (Kalyan); 28. Dr. H. J. Nansey (Jamnagar); 29. Dr. Basudeb Maitra (Howrah); 30. Dr. P. Mukherjea (Howrah); 31. Dr. S. M. Ghosal (Patna); 32. Dr. P. D. Daftary (Ghatkopar); 33. Dr. A. D. Daftary (Bombay); 34. Dr. V. K. Torarkar (Bombay); 35. Dr. K. N. Misra (Cuttack); 26. Dr. A. C. Sen Gupta (Dhakuria); 37. Dr. M. Y. Mirza (Hyderabad); 38. Dr. Gour Hari Dutta (Ranaghat, Bengal); 39. Dr. Saroj Bose (Calcutta); 40 Dr. Sukomal Sen (Calcutta); 41. Dr. R. A. Bhagwat (Indore); 42. Dr. M. N. Surya Prakasa Rao (Chittoor); 43. Dr. Debesh Mukherjee (Calcutta); 44. Dr. M. C. Marulappa (Chitaldrug); 45. Dr. Amarendra Nath Roy (Bengal); 46. Dr. S. M. Chaterjee (Budge Budge); 47. Dr. R. K. Roy (Calcutta); 48. Dr. Mohini Mohan Ray (Kotalpur); 49. Dr. S. L. Gupta (Dewas, Ratlam); 50. Dr. A. N. Apte (Indore); 51. Dr. D. P. Jayaram (Bangalore); 52. Dr. D. B. Goyal (Punjab); 53. Dr. M. A. Hafeez (Bangalore); 54. Dr. Mrinal Nandi (Calcutta); 55. Dr. M. Guru Das (Bangalore); 56. Dr. Y. D. Kapur (Faizabad); 57. Dr. R. N. Gupta (Lucknow); 58. Dr. B. N. Mehrotra (Lucknow); 59. Dr. K Rangacharyulu (Hyderabad); 60. Dr. P. Manikyaraju (Secunderabad); 61. Dr. Sisir Sengupta (Calcutta); 62. Dr. Gopi Krishna Chakravarti (Bagnan); 63. Dr. B. K. Nagar (Ujjain); 64. Dr. S. C. Misra (Cuttack); 65. Dr. D. Das (Bhadrak); 66. Dr. (Capt.) A. K. Nath (Nabadwip); 67. Dr. T. J. Lalwani (Bombay).

Dr. C. O. Karunakaran, the President, took the chair.

I. *Confirmation of the Proceedings of the Annual Meeting of the Central Council held at Cuttack on the 24th and 26th December, 1958.*

The following corrections were made in the Minutes as circulated :

Under the list of Members Present, Serial No. 157, change "Mysore" to "Bangalore".

Serial No. 163, change "Bihar" into "Gadag".

Page No. 4, Item No. 7 (3)—to read as under :—

"The Central Office stated that in response to the Memorandum sent to the Union Minister of Finance, along with a request for an interview, the Association was advised to seek an interview with the Chairman of the Life Insurance Corporation. It was further stated by the Office that such a step had already been taken in the past without any fruitful results".

Page No. 6 : In the list of donations, delete the name of Dr. M. V. Suryaprakash Rao (Serial No. 2) having donated Rs. 50/75 nP.

Page 8.

Under item No. 6, Expenditure side, in the first para, 2nd line, after the word "deleted", add :

"As proposed by Dr. Debesh Mukherjee (Calcutta) and seconded by Dr. Souren Sengupta (Calcutta)".

In the same para of the same item, 2nd line, substitute "Dr. Souren Sen Gupta (Calcutta)" instead of "Dr. Arvind M. Shah (Bombay)" and in the 3rd line of the same para, substitute "Dr. Debesh Mukherjee (Calcutta)" instead of "Dr. S. N. Acharya".

Page 12.

Item No. 12 (II) : In the bracketted sentence, Dr. Debesh Mukherjee (Calcutta), seconded by Dr. S. K. Sen (Calcutta) pointed out that Rule 32 had been omitted in the minutes after the word "Bye-laws". Several speakers opposed this contention and on a vote being taken, 34 members opined that the line as presented was correct and 18 members opined that it was not so.

The President declared that the record was correct.

In the 2nd line of last para of the same item, the word "feels" was decided to be deleted, as suggested by the President.

Page No. 14, under item VI (ii) :

Change "Dr. H. N. Shivapuri (Lucknow)" to "Dr. M. A. Panwala (Bombay)" and "Dr M. M. S. Siddhu (Lucknow)" to "Dr. V. K. Toraskar (Bombay)".

The minutes of the 19th Annual meetings of the Central Council held on 24th and 25th December, 1958, as corrected above, were confirmed, 43 members for and 16 against (Proposed by Dr. H. N. Shivapuri (Lucknow) and seconded by Dr Arvind M. Shah (Bombay).

II. *Delegation of all or some of the powers of the Central Council to the Working Committee, till the next Working Committee is constituted, except the power of altering the Rules.*

The following resolution was proposed by Dr. H. N. Shivapuri (Lucknow) and seconded by Dr. V. P. Vasudevan (Coimbatore) :

"Resolved that the Central Council of the Indian Medical Association delegates to the Working Committee all its powers and functions excepting the power of altering the rules, till the next ordinary meeting of the Council."

The resolution was carried unanimously.

III. *Any other business with the permission of the President.*

(1) Dr. H. N. Shivapuri (Lucknow), seconded by Dr. Arvind M Shah (Bombay) proposed that Bye-laws

7, 17 and 19 as amended at the Annual Meeting of the Central Council, should become applicable from 1st October, 1959. Several members opposed this proposal of Dr. H. N. Shivapuri. On a vote being taken, 34 members supported Dr. Shivapuri, with none opposing.

The proposal was thus carried.

(2) Dr. P. K. Guha, Editor of the Journal, I.M.A., requested that as per the Provident Fund Rules of the Journal Office, the Provident Fund Account at Calcutta should be operated according to the provision for this matter in the Rules.

The Central Council acceded to this request of Dr. Guha.

(3) The following two members donated Rs. 50/- each towards the Building Fund of the I.M.A. from out of their T.A. for the annual meeting of the Central Council held on 24th and 25th December 1958 :—

1. Dr. M. A. Hafeez (Bangalore).
2. Dr. M. C. Marullapa (Chitaldrug Branch).

The meeting then ended with a hearty vote of thanks to the chair.

C. O. KARUNAKARAN
President
Indian Medical Association.

VED PRAKASH
Hony. Joint Secretary
Indian Medical Association.

BRANCH NOTES

AGRA BRANCH—A meeting was held on 7-9-59, Dr. S. P. Srivastava presided. Dr. B. P. Agarwal showed an interesting case of Arterio-Venous Aneurism in the Abdominal Cavity. Dr. R. S. Dayal spoke on Diarrhoea in Infancy. Dr. Dixit also gave his impression about this disease. The meeting desired postponement of the State meeting for a week due to election of Corporations in 5 Kabal towns.

ANANTAPUR DISTRICT BRANCH—The general body meeting of the branch was held on 26-9-59 with Dr. M. P. Sarma in the chair. The annual report and the statement of accounts were adopted. Office-bearers for 1959-60 were elected with Dr. M. P. Sarma as president, Dr. Mrs. N. Narayanamma as vice-president, Dr. V. K. Rajaram Rao as hony. secretary, Dr. K. Rangiah Chetty as joint secretary. Dr. J. Somasundaram read a paper on Peptic Ulcer. Dr. G. Ramiah Chetty presented a case of a young man suffering from Thyrotoxicosis. The membership strength in 1958-59 showed slight improvement. Four ordinary and 1 special meeting were held during the year. In the meetings during 1958-59, Dr. A. Sannappa, Dr. C. R. Naidu, Dr. S. C. Sen, Dr. M. Purushotham Sarma and Dr. J. J. Dharmoraj spoke on subjects of their choice.

BALASORE BRANCH—The last monthly meeting of the branch was held on 18-10-59. Office-bearers for 1959-60 were elected with Dr. A. K. Biswas as president, Dr. B. K. Giri and Dr. R. R. Debi as joint secretaries. The outgoing president pleaded for a healthy relationship between the general practitioner and the patient.

BANARAS BRANCH—The annual report of the branch for the year 1958-59 shows that membership increased from 107 to 117. Twenty general body meetings excluding the annual general meeting were held during the year. At 11 of these meetings scientific subjects were discussed. At 3 other meetings, scientific films were shown. Many prominent medical men from outside the State spoke in some of the meetings. Interesting films were also shown in some of the meetings. The attendance at the meetings was good. The Council of the branch held 10 meetings. Apka-Swasthya, the monthly magazine published from this branch made steady progress. As in previods years special Children's Holi, School Aid and World Health Numbers were brought out.

BIJNOR BRANCH—The annual report for 1957-58 shows that membership position remained unchanged. Eight clinical, 2 emergent and 8 regular meetings were held during the year. The World Health Day was celebrated on 7-4-59. A symposium on Mental Hygiene was held on that date. The 2nd Bijnor Medical Conference was held on 18-11-59. Four journals were being subscribed during the year.

BULANDSHAHR BRANCH—The annual report of the branch for 1958-59 shows that 10 meetings were held during the year. Five interesting papers were read at the meetings. Membership increased by 6. The branch contributed Rs. 240/- towards the Building Fund, Central Office, IMA.

DARBHANGA BRANCH—The annual general meeting was held on 27-9-59. The annual report, statement of accounts and auditor's report were presented and adopted. A condolence resolution on the death of Dr. B. N. Banerjee was passed. The following office-bearers were elected : President—Dr. Baldeo Prasad Singh; Vice-presidents—Dr. S. N. Sinha; Dr. T. C. Gupta; General secretary—Dr. Hari Prasad; Jt. Secretary—Dr. B. B. P. Roy; Propaganda secretary—Dr. K. P. Agarwal; Treasurer—Dr. S. M. Mishra; Auditor—Dr. Purushottam Prasad Singh. Dr. S. Hoda, K. P. Agarwal and Dr. S. N. Sinha were elected to the State Council.

Several resolutions were passed one of which stated that at least 4 clinical meetings should be held annually.

DAVANGERE BRANCH—The annual general meeting was held on 27-9-59. Office-bearers for 1959-60 were elected with Dr. P. Naganagowda as president, Dr. Dhananjaya as vice-president and Dr. G. Gopal as hony. secretary.

ERNAKULAM-COCHIN BRANCH—A meeting of the branch was held on 16-9-59. Office-bearers for 1959-60 were elected with Dr. M. J. Philip as president; Dr. S. Vasava Menon, Dr. V. Krishnankutty and Dr. (Mrs.) Chandramathy Amma as vice-presidents; Dr. M. A. Urumese as hony. secretary; Dr. Mohamed Balu, Dr. (Mrs.) Mary Antony as hony. joint secretaries; Dr. V. J. Paul as hony. treaturer; Dr. N. Vasudevan as member, Central Council.

FAIZABAD BRANCH—The annual report of the branch for 1958-59 shows that 12 meetings including annual meetings were held during the year. In the scientific section 4 papers were read and 5 case notes demonstrated and discussed. Interesting films were also shown. Six new members were enlisted during the year. Three journals were subscribed and books purchased for the library. A sum of Rs. 25/- was contributed to the Assam Flood Relief Fund.

The annual meeting of the branch was held on 11-9-59. Dr. Y. D. Kapur presided. Office-bearers for 1959-60 were elected with Dr. Pratap Bahadur as president; Dr. Y. D. Kapur as hony. secretary. He was also elected on the Central Council, IMA. The annual dinner was held after the elections.

A special meeting was held to welcome Dr. K. M. Lal, Director, Medical and Health Services, U.P. Dr. Y. D. Kapur presided. Dr. Lal spoke on Mental Health and the importance it is receiving in the third Five Year Plan. A special dinner was held after the meeting.

FARRUKHABAD - CUM - FATEGARH BRANCH—At the annual general meeting held on 30-10-59, the following office-bearers for 1959-60 were elected : President—Dr. Mrs. C. H. Lal; Vice-Presidents—Dr. P. N. Vatsal, Dr. C. V. Chaturvedi; Hony. Secretary and Treasurer—Dr. Jagat Narain Saksena; Joint Secretary—Dr. P. S. Sud; Dr. Mrs. C. H. Lal and Dr. Jagat Narain Saksena were elected to the State Council, while Dr. S. D. Hajela was elected to the Central Council with Dr. K. N. Tandon as alternate.

Dr. Bhola Nath, President-elect, U. P. Medical Conference, addressed the meeting.

FORBESGANJ BRANCH—The annual general meeting of the branch was held on 4-10-59. Office-bearers for 1959-60 were elected with Dr. A. Niranjan as president, Dr. P. N. Roy as vice-president, Dr. B. K. Sarkar as hony. secretary, Dr. Samsamul Huque as joint secretary and Dr. A. K. Chatterjee (alternate, Dr. M. Ahmed) as Central Council representative.

GHUGUDANGA BRANCH—The monthly meeting of the branch was held on 27-9-59. Dr. K. K. Dutta presided. Seven members were present. The enhancement of C.F.C. from Rs. 8/- to Rs. 9/- per annum was discussed. The annual meeting of the branch was postponed till November.

GIRIDIH BRANCH—The annual general meeting was held on 30-9-59 with Dr. H. Chatterji in the chair. After the secretary had presented his report the following office-bearers were elected: President—Dr. H. Chatterji; Vice-presidents—Dr. A. P. Roy, Dr. G. Sahay; Secretary—Dr. T. Ahmad; Jt. Secretary—Dr. S. Choudhury.

The annual subscription was raised. Circulars received from the State and the Central were read and adopted. Members agreed to pay their quota for the Central Building Fund. The meeting came to a close with a dinner.

HOWRAH BRANCH—The annual general meeting of the branch was held on 25-10-59. Office-bearers for 1959-60 were elected with Dr. N. N. Sinha as president; Dr. R. Halder, Dr. B. Chowdhury and Dr. B. Mukherjee as vice-presidents; Dr. D. P. Mukherjee as hony. secretary; Dr. B. Maitra, Dr. D. Malik and Dr. D. Dutta as hony. joint secretaries; Dr. D. P. Mukherjee and Dr. B. Moitra, (alternate members; Dr. B. Das and Dr. D. Dutta) as representatives to the Central Council. The annual report for 1958-59 shows that 132 members were on the roll. Two clinical meetings were held; Dr. S. Dutta spoke on Common Difficulties in our everyday practice in the eye of a specialist. Dr. Major H. K. Indra spoke on Common Ophthalmic Condition in general practice. Five executive meetings were held. In addition to the annual general meeting 4 special general meetings were held.

JAHANABAD BRANCH—The annual meeting was held on 25-10-59 under the presidentship of Dr. R. Saran. The following office-bearers for 1959-60 were elected: President—Dr. R. Saran; Vice-president—Dr. N. G. Sinha; Hony. Secretary and Treasurer—Dr. B. N. Goswami; Hony. Jt. Secretary—Dr. Jogendra Prasad; Public Relations Officer—Dr. S. D. Mishra; Organising Secretary—Dr. B. Rai Choudhury. The annual report was read by the secretary and unanimously adopted.

It was decided that the State Branch should recommend to the Government that the services of the medical officers of the district board, who are all being absorbed in Government service as fresh appointments, should be countered.

JAMTARA BRANCH—At the annual meeting (date not mentioned) under the chairmanship of Dr. G. S. P. Sinha, with 11 members attending, the following office-bearers, for 1959-60 were elected. President—Dr. G. S. P. Sinha; Vice-President—Dr. S. P. Ghosh; Secretary—Dr. S. K. Gon; Treasurer—Dr. P. K. Gon; Drs. G. S. P. Sinha and R. N. Sarkar were elected to the State Council and Dr. S. K. Gon to the Central Council with Dr. P. K. Mukherjee as alternate. It was resolved to increase the annual subscription from Rs. 12/- to Rs. 13. Several resolutions were sent to be moved in the State Council meeting.

The annual report says that the branch was started with 6 members and had now 25 on its rolls. There were 4 meetings during the year.

KADI-KALOL BRANCH—The annual report of the branch shows that there were 6 meetings during the year. Various scientific subjects were discussed in the meetings. Office-bearers for the year 1959-60 were elected with Dr. M. C. Patel as president, Dr. P. R. Patel as vice-president, Dr. V. V. Kapadia as treasurer, Dr. H. N. Trivedi as secretary and Dr. M. C. Shah as joint secretary.

KARIMNAGAR DISTRICT BRANCH—A clinical meeting was held on 25-10-59. Dr. K. N. Vaghray presented a case of Face Presentation with Cord Round the Neck delivered by caesarian and Dr. Srinivasan presented another case of Septic Meningitis. Dr. Jagan Mohan presented a case of Lipoma. Dr. Mohd. Gulam Nabi described two interesting cases.

The members were entertained to dinner by Dr. Krishna Rao and Dr. M. Fakruddin Khan.

LOHARDAGA DISTRICT BRANCH—The general meeting was held on 21-10-59, Right members were present. Resolutions were passed appreciating the formation of legal defence union, recommending the exploration of the possibility of a new medical college at Patna or elsewhere, endorsing the pay scales as suggested by the Bihar State Branch, appreciating the recommendation of the IMA to give importance to health work in the third Plan.

MADHIPURA BRANCH—An ordinary meeting was held on 25-9-59, with 11 members present. Dr. R. P. Singh presided. Accounts from 1-6-59 to 24-9-59 were placed before the meeting. In a resolution it was decided to raise the annual subscription from Rs. 10 to Rs. 11. A sub-committee of 5 members and 4 prominent members of the public was formed to find ways and means to increase the funds of the branch.

Later a special meeting was held, presided over by Dr. B. D. Ganguly. The house elected Dr. Ramanand Prasad Singh as President of the branch in place of Dr. S. S. Prasad who had been transferred.

MADURA BRANCH—A monthly meeting was held on 26-9-59, Dr. K. Gopal presiding. A condolence resolution was passed mourning the death of Dr. A. Narayan Menon, ex-secretary, ex-president and life member of the branch.

It was decided to invite the XIV Madras State Medical Conference to be held in Madurai in January 1960.

Dr. S. Perumal Raian gave an interesting talk on Dental Surgery in General Practice.

A monthly meeting was held 24-10-59 under the presidentship of Dr. K. Gopal. References were made by several members about the Late Dr. A. Narayana Menon, one of the founder-members of the branch. Election of office-bearers for the Reception Committee and other sub-committees for the XIV Madras State Medical Conference to be held in Madurai in January 1960, were conducted.

Dr. S. A. Kabir gave a lecture on Modern Trends in Anaesthesia.

MADRAS CITY BRANCH—A monthly clinical meeting was held on 26-9-59, with Dr. G. Sriramulu in the chair, 265 members being present.

At the outset a condolence resolution was passed on the death of Dr. R. K. Thampan. This was followed by a lecture by Dr. R. Subramaniam on the Management of Essential Hypertension, illustrated with slides.

Aftr the meeting a dinner was arranged to welcome new members, who were addressed by the President Dr. Sriramulu and then by Dr. V. Venkappa, ex-President, I.M.A.

Dr. K. S. Sanjivi later spoke on Voluntary Health Service.

Mr. N. K. Chandra of Standard Pharmaceutical Works who was the evening's host also spoke.

MADRAS STATE BRANCH—The results of the election of President and vice-presidents were announced at a meeting held on 19-9-59. These are: President—Dr. T. V. Sivanandan; Vice-presidents—Dr. A. G. Leelakrishnan (senior) and Dr. C. Nathamuni Naidu.

MUZAFFARPUR BRANCH—The annual general meeting of the branch was held on 5-10-59. Twentyeight members were present. The members condoled the death of Dr. P. A. Singh, D.H.S. Behar. The annual report for 1958-59 was read and adopted. Office-bearers for 1959-60 were elected with Dr. R. Sinha as president, Dr. G. Prasad, Dr. G. S. Mujtaha, Dr. Miss R. S. Bakla as vice-presidents, Dr. G. P. Seth as hony. secretary,

Dr. S. K. Sidhanta as hony. joint secretary, Dr. P. K. Sinha as hony. treasurer.

NAGERCOIL BRANCH—The annual meeting of the branch was held on 19-9-59. Office-bearers for 1959-60 were elected with Dr. M. Mathias as president, Dr. S. Muthukaruppa Pillai as vice-president, Dr. D. Jayaharan as hony. secretary, Dr. M. Gopala Pillai as hony. treasurer.

SANGLI BRANCH—The annual meeting of the branch was held on 18-10-59. Dr. D. V. Desai presided. The annual report was read by the secretary and passed. Office-bearers for 1959-60 were elected with Dr. M. A. Paranjape as president and Dr. R. N. Khanapurkar as hony. secretary. Dr. K. P. Bapat was felicitated on his 75th birthday. Twentysix members were present.

SHOLAPUR BRANCH—The annual report of the branch shows that membership during 1958-59 increased from 82 to 101. The following meetings were held during the year: (1) Clinical and business combined general body meetings—7; Purely Clinical meeting—1; Purely business meeting—1; Special general body meetings—10; executive body meetings—7; provincial council meetings—2; Central Council meeting—1; V. M. U. C. meetings—4; Address by guests—7; Farewell meeting—1; Among the extraordinary activities of the branch may be mentioned, (1) holding of Inter-District Conference, (2) Formation of an ad-hoc committee for trying for the establishment of a medical college at Sholapur etc. Dr. Bland of WHO, Dr. Wardekar, Dr. Subramanian and some other eminent guests spoke to the members during the year on their respective subjects. The clinical meetings were held in the Rly. hospital and Seth S. N. Hospital. Eleven periodicals were subscribed to and 12 new books were purchased during the year. The World Health Day was celebrated as usual. The branch donated substantial amounts to various funds. A need for a centrally placed hall for holding meetings other than clinical was felt and with the consent of the superintendent of the H. D. High School, meetings were being held in the Kala Vibhag of the school and the library also had been located there. The Employees' State Insurance Scheme sub-committtee held 1 meeting during the year. The report of the building sub-committee shows that a plot for building would be available soon. The annual report of the Schools sub-committee sltows that total number of students examined were 3637. The annual report of the representative of I.M.A. on the Hind Kustha Nibaran Sangha, Sholapur, says that the work of the institution was being carried efficiently under the supervision of the Medical Officer-incharge. The report of the representative of the branch on the Dufferin Committee contained certain recommendations for equipment in the Labour Theatre. The total number of trips made by the members of the Village Medical Uplift Sub-Committee was 102. The total number of patients attended to was 878. Eighty cases were demonstrated at the clinical meetings of the branch during the year. Office-bearers for the year 1959-60 were elected with Dr. L. B. Shrikhande as president, Dr. V. P. Aroskar as vice-president, Dr. P. S. Deshpande as hony. secretary, Dr. M. G. Kirleskar as hony. treasurer.

SULTANPUR BRANCH—The annual general meetin of the branch was held on 6-9-59. Dr. H. C. Varma presided. Three interesting clinical cases were demonstrated by Dr. H. C. Varma and Dr. H. D. Shukla. A review of the Place of Pneumoperitoneum in the Treatment of Pulmonary Tuberculosis was given by Dr. R. Prasad. Office-bearers for 1959-60 were elected with Dr. H. C. Varma as president, Dr. M. M. N. Mathur as vice-president, Dr. J. K. Agrawal as hony. secretary.

TANJORE DISTRICT BRANCH—A monthly meeting was held on 30-8-59. Dr. T. M. Pilay, Vice-President presided. It was decided to raise the annual subscription from Rs. 14 to Rs. 15. Election of Vice-Presidents of the State Branch was held. Dr. N. R. Subramanyam, President of the Branch, spoke on The Management of Heart Failure.

The 23rd annual day was celebrated on 18-10-59. Ninety membrs were present. The president, Dr. Subramanyam presided. After the welcome address by the president, the secretary read the annual report and presented the annual accounts. Dr. P. M. Palani spoke on Medical Emergencies and their Treatment. Dr. H. T. Vira Reddy spoke on Abdominal Emergencies in Surgical Practice and exhibited some slides.

The following office-bearers were elected at the business meeting: President—Dr. N. R. Subramanyam, Vice-President—Dr. T. M. Pillay; Hony. Secretary and Treasurer—Dr. S. Narayanan.

Dr. T. M. Pillay, V. C. Gupta, T. R. Ramachandran and P. Narayana Rao were elected to the State Council.

Later a symposium on Some Aspects of Chest Diseases—Medical and Surgical was held. Participants were: Dr. A. L. Annamalai, who spoke on medical aspects and Dr. M. S. Sadasivan who spoke on the surgical aspects.

TARAKESWAR BRANCH—A general meeting of the branch was held on 17-5-59. Twenty members were present. Dr. K. Das presided. In the opinion of the members, the age limit of medical officers for retirement should be raised to 58. Dr. H. S. Datta, vice-president, spoke on child psychology.

A general meeting was held on 30-8-59. Twenty-six members were present. Farewell as given to Dr. S. P. Roychowdhury on his retirement from service. It was decided that while C.F.C. was increased from Rs. 8/- to Rs. 9/-, local fund contribution should be curtailed by Re. 1/-. Dr. B. Das spoke on Hormones in Gynaecological Practice based on Statistics of Chittaranjan Seva Sadan.

TENALI BRANCH—At the annual meeting held on 26-9-59 with Dr. G. Satyanarayana Rao in the chair, the following officer-bearers for 1959-60 were elected: President—Dr. C. Krishnaiah; Vice-President—Dr. J. Venkateswara Rao; Secretary—Dr. K. Punnaiah, Jt. Secretary—Dr. C. V. Subbarao.

TIRUCHIRAPALLI DISTRICT BRANCH—At the annual meeting held on 25-10-59, the following office-bearers for 1959-60 were elected: President—Dr. G. Viswanathan; Vice-President—Dr. P. V. Sundaran; Hony. Secretary—Dr. V. K. Ranganathan; Jt. Secretary and Treasurer- Dr. M. V. Iswaran; Representatives to State Council: Dr. V. Ananthagiri, Dr. T. S. Balasubramanyam, Dr. R. Kalamegham, Dr. N. C. Subramanyam, Dr. R. Sambasivan, Dr. S. Venkatesan; Representatives to Central Council: Dr. T. S. Balasubramanyam and Dr. T. V. Srinivasan.

TIRUNELVELI DISTRICT BRANCH—The annual meeting of the branch was held on 26-9-59. Office-bearers for 1959-60 were elected with Dr. P. Ragabendra Acharya as president, Dr. S. Maragathavel as vice-president, Dr. P. Vadirale as hony. secretary and Dr. K. Ramchandra Naidu as member of the Central Council.

UDIPI BRANCH—The annual meeting of the branch was held on 18-10-59 with Dr. S. R. Pai in the chair. Office-bearers for 1959-60 were elected with Dr. S. R. Pai as president, Dr. P. R. Nayak as vice-president, Dr. V. L. Pandit as hony. secretary, Dr. K. Mohandas Pai as joint secretary. The report for 1959-60 was recorded. Dr. K. Rajagopal of Kasturba Medical College gave a talk on the Recent Advances in the Field of Nutrition.

ULUBERIA BRANCH—The annual general meeting of the branch was held 2-10-59. The hony. secretary reported that the members of Fuleswar Branch closed down, joined the Uluberia branch. The present membership is 31. Office-bearers for 1959-60 were elected with Dr. K. L. Basu Mallik as president, Dr. K. P. Mitra as vice-president, Dr. P. Chatterjee as hony. secretary, Dr. P. C. Sarker as joint hony. secretary.

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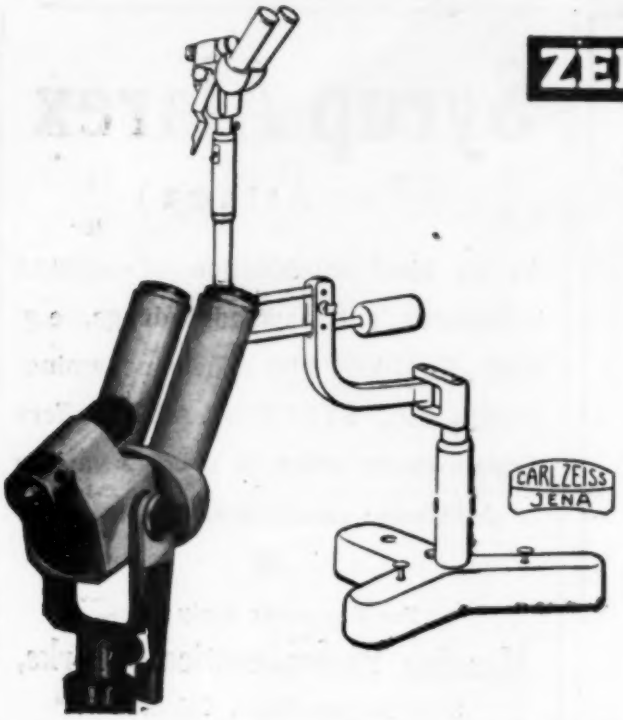

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(Lancet, 1958. 1525)

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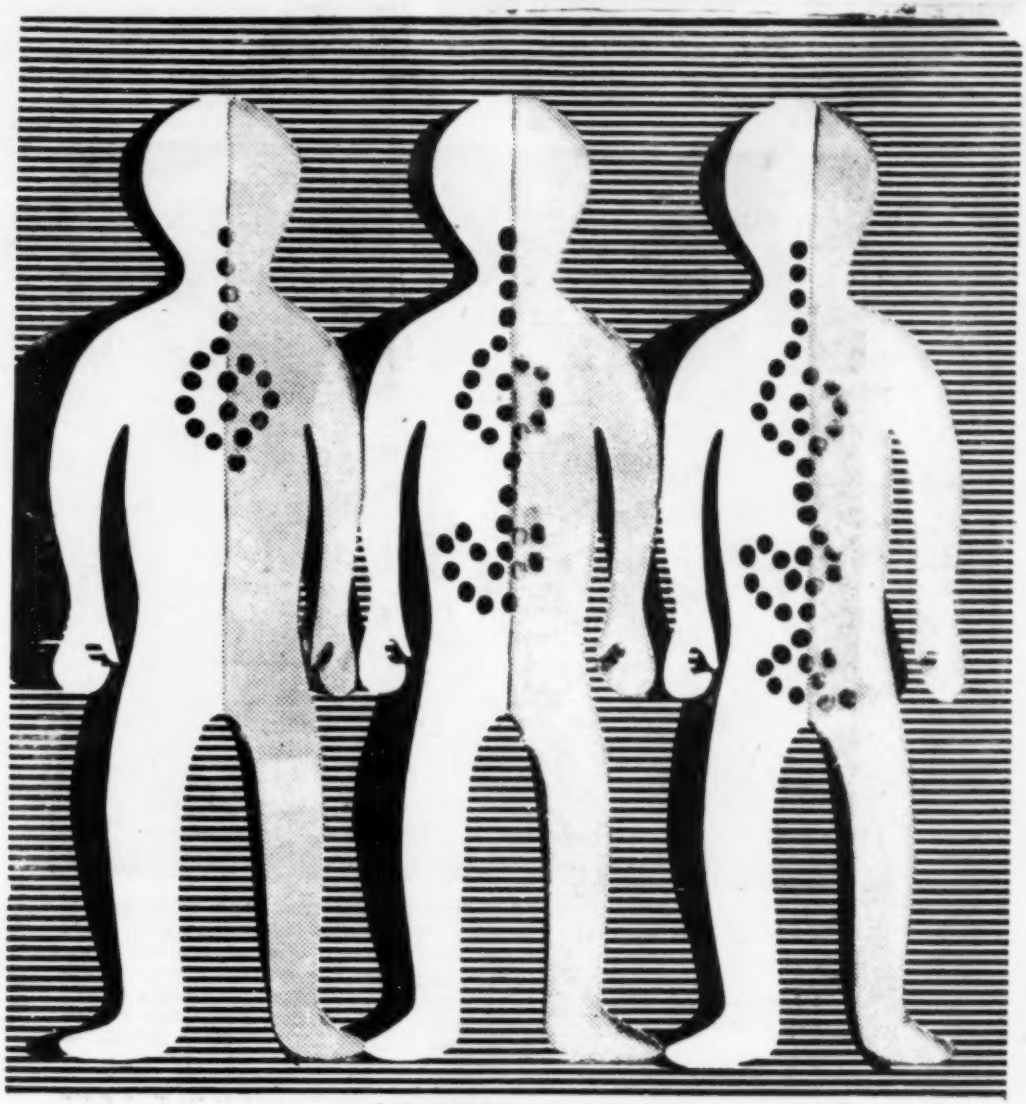

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1. *Lamphier, T.A.: Ann. N.Y. Acad. of Sc., 67:10, p. 810, May 1957.*

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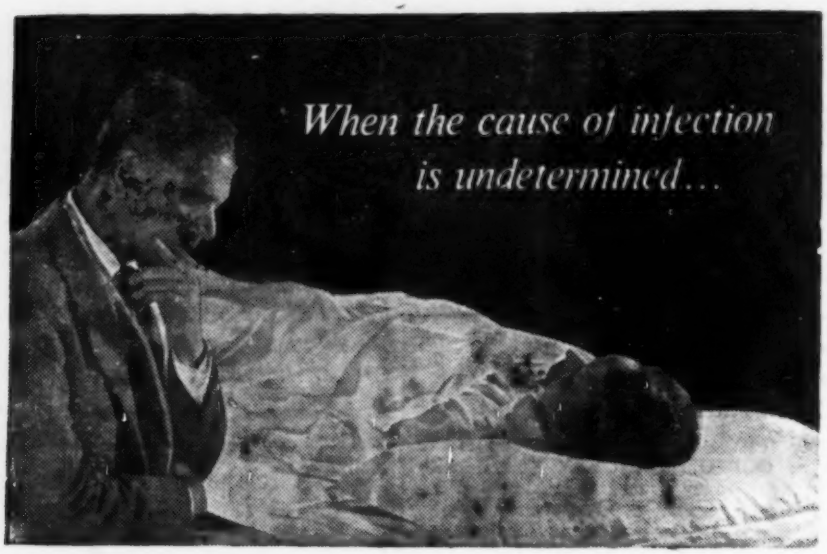
Editor—Dr. P. K. Guha, M.B., M.R.C.S. (Eng.), D.O.M.S. (Lond.)



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Cover and Advertisement pages printed at the Venus Printing Works, 52/7, Bipin Behari Ganguly Street, Calcutta-12.